A Dissertation on

EVALUATION OF BRAINSTEM AUDITORY EVOKED POTENTIAL IN ESSENTIAL HYPERTENSION

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Branch - V : M.D. (Physiology)

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CERTIFICATE

This is to certify that **"EVALUATION OF BRAINSTEM AUDITORY EVOKED POTENTIAL IN ESSENTIAL HYPERTENSION''** is bonafide work done by the Post Graduate Dr.PRIYA .V, Department of Physiology, Kilpauk Medical College, Chennai – 10 under my guidance and supervision in partialfulfilment of regulations of **The Tamilnadu Dr.M.G.R.Medical University** for the award of M.D (PHYSIOLOGY) BRANCH – V during the academic period from 2012 – 2015.

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DECLARATION

I**, DR.V.PRIYA**, solemnly declare that dissertation titled "**EVALUATION OF BRAINSTEM AUDITORY EVOKED POTENTIAL IN ESSENTIAL HYPERTENSION"** is a bonafide work done by me at Institute of Physiology,Kilpauk Medical College and Hospital from August 2013 to July 2014 under the guidance and supervision of **Prof.Dr.A.RASHEEDA**, M.D., Professor of Physiology. This dissertation is submitted to The Tamilnadu Dr.M.G.R.Medical University ,towards partial fulfilement of requirement for the award of **M.D.Degree (Branch – V**) in Physiology.

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LIST OF ABBREVIATIONS

BAEP	–	Brainstem Auditory Evoked Potentials
HT	–	Hypertension
CHD	–	Coronary Heart Disease
ABR	–	Auditory Brainstem Response
MRFIT	–	Multiple Risk Factor Intervention Trial
PRA	–	Plasma Renin Activity
PIH	–	Pregnancy Induced Hypertension
mA	–	milli – Amperes
mV	–	millivolts
mΩ	-	milli – ohms
ms	–	milliseconds
µS	–	microseconds
ADC	–	Analogue to Digital Converter
IPL	–	Inter Peak Latencies
Ai	–	Ipsilateral Ear
AC	–	Contralateral Ear
CRO	–	Cathode Ray Oscilloscope
APL	-	Absolute Peak Latency

ABSTRACT

Central nervous system dysfunctions are common in patients of essential hypertension.Arterial and arteriolar spasm in cerebral blood vessels may be responsible.Micro-infarctions are known to be responsible for dysfunction in heart,kidney ,brain and other tissues in patients of hypertension.Brainstem auditory evoked potentials are the potentials recorded from the ear &vertex in response to a brief auditory stimulation to assess the conduction through auditory pathway upto midbrain.

This study will be executed to evaluate subclinical brainstem auditory evoked potentials abnormalities in essential hypertension.

- **STUDY DESIGN :**

  -Case control study**.**

INTRODUCTION

Blood pressure is the lateral pressure exerted by the flowing blood on the walls of the vessels[5].It is usually measured in mmHg. Without any further qualification the term blood pressure denotes arterial pressure. While describing the pressure exerted by the blood column in other types of blood vessels, the type of vessels is also mentioned , e.g. capillary pressure and venous pressure.

Very few diseases are responsible for frequent and severe complications due to Arterial Hypertension[8]. Insufficiency of blood flow to Heart, Kidney and peripheral blood vessels is common in arterial hypertension[10, 11]. Moreover, it is estimated that about half of the deaths of patients above 50 years are due to cardiovascular diseases, and 80% of them have high blood pressure. Human body depends on proper supply of oxygen and nutrients [10] in order to maintain their function and such supply depends on functional and structural integrity of heart and blood vessels. High pressure in vascular system may cause haemorrhage in inner ear supplied by anterior inferior cerebellar artery[14] .This artery supports the inner ear, cochlear artery and anterior vestibular artery which may cause progressive or sudden hearing loss.[8]

Hypertension is an accelerating factor for degeneration of hearing apparatus[55].In patients with Primary Hypertension ,Central nervous system dysfunctions occurs frequently[1].Systemic arterial hypertension is

an independent risk factor for hearing loss [8].Regulation of blood pressure at the brainstem level is affected in Essential Hypertension. Arterial and arteriolar spasm along with fibrinoid degeneration leads to microinfarction and cerebral edema in severe cases of hypertension[30]. Hence ,the sensory deficit could be due to either the cause i.e primary disorder of sympathetic system responsible for essential hypertension or due to it's effects.[22]

As per Bernoulli's experiment ,the lateral pressure exerted on the vessel by the flowing blood represents the potential energy and the end on pressure represents the total energy, i.e kinetic energy plus potential energy[5].Kinetic energy depends on the velocity of blood flow. Therefore, velocity of flowing blood remaining constant, a rise in lateral pressure indicates a rise in perfusion pressure and vice versa.[5]

HYPERTENSION

Definition: Hypertension (HT) refers to a condition in which value of systolic blood pressure is persistently more than 140mmHg and /or that of diastolic blood pressure is above 90mmHg[5] .If there is increase only in systolic blood pressure ,it is called systolic hypertension in which pulse pressure is raised. In adults there is a continuous, incremental risk of cardiovascular disease, stroke, and renal disease across levels of both systolic and diastolic blood pressure. The Multiple Risk Factor Intervention Trial (MRFIT), which included >350,000 male participants,

demonstrated a continuous and graded influence of both systolic and diastolic blood pressure on CHD mortality, extending down to systolic blood pressures of 120 mmHg. Similarly, results of a meta-analysis involving almost 1 million participants indicate that ischemic heart disease mortality, stroke mortality, and mortality from other vascular causes are directly related to the height of the blood pressure, beginning at 114/76 mmHg, without evidence of a threshold. Cardiovascular disease risk doubles for every 20-mmHg increase in systolic and 10-mmHg increase in diastolic pressure[4]. Among older individuals, systolic blood pressure and pulse pressure are more powerful predictors of cardiovascular disease than diastolic blood pressure.

Clinically, hypertension might be defined as that level of blood pressure at which the institution of therapy reduces blood pressure–related morbidity and mortality. Current clinical criteria for defining hypertension are generally based on the average of two or more seated blood pressure readings during each of two or more outpatient visits. A recent classification recommends blood pressure criteria for defining normal blood pressure, prehypertension, hypertension (stages I and II), and isolated systolic hypertension, which is a common occurrence among the elderly[4]. In children and adolescents, hypertension is generally defined as systolic and/or diastolic blood pressure consistently >95th percentile for age, gender, and height.

Blood Pressure Classification		
Blood Pressure Classification	**Systolic, mmHg**	**Diastolic, mmHg**
Normal	<120	*and*<80
Prehypertension	120–139	*or* 80–89
Stage 1 hypertension	140–159	*or* 90–99
Stage 2 hypertension	160	*or* 100
Isolated systolic hypertension	140	*and*<90

Home blood pressure and average 24-h ambulatory blood pressure measurements are generally lower than clinic blood pressures. Because ambulatory blood pressure[4] recordings yield multiple readings throughout the day and night, they provide a more comprehensive assessment of the vascular burden of hypertension than a limited number of office readings. Increasing evidence suggests that home blood pressures, including 24-h blood pressure recordings, more reliably predict target organ damage than office blood pressures. Blood pressure tends to be higher in the early morning hours, soon after waking, than at other times of day. Myocardial infarction and stroke are more frequent in the early morning hours. Nighttime blood pressures are generally 10–20% lower than daytime blood pressures, and an attenuated nighttime blood pressure "dip" is

associated with increased cardiovascular disease risk. Blunting of the day-night blood pressure pattern occurs in several clinical conditions, including sleep apnea and autonomic neuropathy, and in certain populations, including African Americans[4]. Recommended criteria for a diagnosis of hypertension are average awake blood pressure 134/86 mmHg and asleep blood pressure 120/76 mmHg. These levels approximate a clinic blood pressure of 140/90 mmHg.[4]

Approximately 15–20% of patients with stage 1 hypertension based on blood pressures have average ambulatory readings <134/86 mmHg. This phenomenon, so-called white coat hypertension, may also be associated with an increased risk of target organ damage (e.g., left ventricular hypertrophy, carotid atherosclerosis, overall cardiovascular morbidity), although to a lesser extent than individuals with elevated office and ambulatory readings. Individuals with white coat hypertension are also at increased risk for developing sustained hypertension.

Types of hypertension: It is of two types

1. Primary hypertension
2. Secondary hypertension

Essential Hypertension

Essential hypertension tends to be familial and is likely to be the consequence of an interaction between environmental and genetic factors.

The prevalence of essential hypertension increases with age, and individuals with relatively high blood pressures at younger ages are at increased risk[4] for the subsequent development of hypertension. It is likely that essential hypertension represents a spectrum of disorders with different underlying pathophysiologies. In the majority of patients with established hypertension, peripheral resistance is increased and cardiac output is normal or decreased; however, in younger patients with mild or labile hypertension, cardiac output may be increased and peripheral resistance may be normal.

When plasma renin activity (PRA) is plotted against 24-h sodium excretion, ~10–15% of hypertensive patients have high PRA and 25% have low PRA[4]. High-renin patients may have a vasoconstrictor form of hypertension, whereas low-renin patients may have a volume-dependent hypertension. Inconsistent associations between plasma aldosterone and blood pressure have been described in patients with essential hypertension. The association between aldosterone and blood pressure is more striking in African Americans, and PRA tends to be low in hypertensive African Americans. This raises the possibility that subtle increases of aldosterone may contribute to hypertension in at least some groups of patients who do not have overt primary aldosteronism. Furthermore, spironolactone, an aldosterone antagonist, may be a particularly effective antihypertensive agent for some patients with

essential hypertension, including some patients with "drug-resistant" hypertension.

Secondary hypertension refers to a condition in which blood pressure is raised due to some other underlying disease.Common causes of secondary hypertension are:

Cardio vascular diseases producing secondary hypertension are atherosclerosis and coarctation of aorta[4] .

Renal diseases causing secondary hypertension are stenosis of renal artery, glomerulonephritis and tumour of juxtaglomerular cells leading to formation of excess of angiotension II.

Endocrinal disorders associated with secondary hypertension are hyperaldosteronism, pheochromocytoma and cushing's syndrome .

Neurological disorders which may produce secondary hypertension include raised intra cranial pressure, tractussolitarious and sectioning of nerve fibres from carotid sinus.

Pregnancy –induced hypertension (PIH) is noticed in some of the pregnant women. Its exact cause is not known. It may be because of some autoimmune processes during pregnancy or the release of some vasoconstrictor agents from placenta or due to excessive secretion of hormones causing rise in blood pressure.

Measurement of Blood pressure

DIRECT METHOD

In animals, cannula or T-tube is inserted into an artery and connected to Mercury manometer and pressure is recorded on the kymograph, or Pressure transducer (strain gauge) which in turn is connected to polyrite for recording.

INDIRECT METHOD

In human beings blood pressure is measured indirectly by using sphygmomanometer.

Sphygmomanometer

Commonly called blood pressure apparatus, it is the instrument used to measure blood pressure[5].It consists of following parts:

1. Manometer. Two types of manometers are used, Aneroid manometer in which metal bellows, mechanical rings and a dial replaces the glass tube mercury manometer. It is also very commonly used nowadays. However, it needs calibration against a mercury manometer from time to time.

Mercury manometer is commonly used in classical sphygmomanometer[5].It consists of a graduated narrow glass tube having

markings 0 to 300mmHg.Upper end of the tube is closed and lower end is connected to an inflatable rubber bag through a rubber tube.

2. The cuff. The blood pressure apparatus cuff also known as ‘armlet’ or ‘Riva-Rocci cuff’ after the name of discoverer) consists of an inflatable rubber bag which is enclosed in a cotton bag having a long strip of inelastic cloth. The dimension of the commonly used rubber bag are 24cm x 12cm.It should be atleast as wide as half the upper arm’s circumference.T he bag width (12 cm) should be more in obese adults ,about 4-5 cm for children, and 2-3 cm for the newborns[5].

3. Air pump. It is a rubber bulb with a one –way valve at its free end, and a ‘leaky valve’ and a knurled screw at the other end where the rubber tube leading to the cuff is attached. The cuff can be inflated by turning the leak-valve screw clockwise and alternatively compressing and releasing the bulb. Deflation is achieved by turning the screw anticlockwise.

Procedure

The blood pressure may be tested with subject lying supine or sitting, but should be physically and mentally relaxed and free from

excitation .The cuff of the blood pressure apparatus is applied on the upper arm with the centre of the rubber bag lying over the brachial artery which lies medially, and its lower edge should be about 3cm above the elbow.

The blood pressure can be measured using palpatory method, oscillatory method or auscultatory method[5].

1. Palpatory method

Palpatory method described by Riva-Roci in 1896,includes following steps:

Palpate and feel pulsations of the radial artery with the tips of finger of left hand.

Keeping fingers of left hand on the pulse ,slowly inflate the cuff using air pump with the right hand until the pulse disappears. Then raise the pressure further by 30 - 40 mm Hg in the manometer[5].

Open the leak valve of air pump and control it so that the pressure gradually falls in steps of 2-4 mm Hg. Note the reading at which pulse just reappears. This reading corresponds to systolic blood pressure .After this deflate the cuff quickly to zero pressure.

Take three readings in each arm, fully deflating the cuff a few minutes between each reading .Mean of the three readings should be

taken as systolic blood pressure. The actual pressure is usually 4-6 mm Hg higher than the recorded value, since initial 2-3 beats being thin are often missed while feeling the pulse.

Disadvantage of thepalpatory method is that the diastolic blood pressure cannot be measured by this method.

2. Oscillatory method

In this method, initial steps upto raising the pressure in the cuff by 30-40 mmHg after the pulse disappears are same.

Then the mercury column in the graduated glass tube of manometer is carefully watched while slowly lowering the cuff pressure. The oscillations will appear and become prominent once the pressure in the cuff is roughly equal to the systolic pressure.

3. Auscultatory method

Auscultatory method described by Korotkoff in 1905[5], is the most useful technique.

Initial steps upto raising the pressure in the cuff by 30-40 mmHg above the level where the radial artery pulse disappears are similar to palpatory method. However, be pre inflating the cuff, the point just before the brachial artery bifurcates in the cubital space, just medial to the tendon of biceps muscle, is marked.

Then diaphragm of stethoscope is placed on the mark in cubital space and is kept in position with the help of thumb and fingers of left hand.[5]

Pressure in the cuff is lowered slowly by opening the leak valve of the air pump with right hand. While doing so, initially no sound is heard . However , when mercury column is lowered further a tap sound is heard. The character and quality of sound goes on changing while further lowering the mercury column by deflating the cuff, and ultimately the sound disappears. These sounds are called **Korotkoff sounds** and from these the levels of systolic and diastolic blood pressure are noted as described below, when the sounds disappear the cuff should be deflated quickly.

Korotkoff Sounds

Phases of Korotkoff sounds:

Phase I sounds start with a **clear tap** which indicates the systolic blood pressure. The clear, tapping and sharp sounds last for 10-12 mmHg fall in mercury column[5].

Phase II sounds are **murmurish**, ie soft and swishing and last for next 14-15 mmHg fall in mercury column.

Phase III sounds are clear**, knocking and banging** in character and last for next 14-15 mmHg fall in mercury column.

Phase V is labelled when **no sound** is heard. Since the beginners may not appreciate beginning of muffling of sounds and therefore ,disappearance of the sound may be considered as mark of diastolic pressure. However, in some clinical situations such as hyperthyroidism and aortic valve insufficiency where the sounds continue to be heard even when the pressure is low, the level at which muffling of sounds starts is to be taken as diastolic blood pressure.

PHYSIOLOGIC ANATOMY OF EAR:

Fig :1Schematic Diagram of Anatomy of Ear

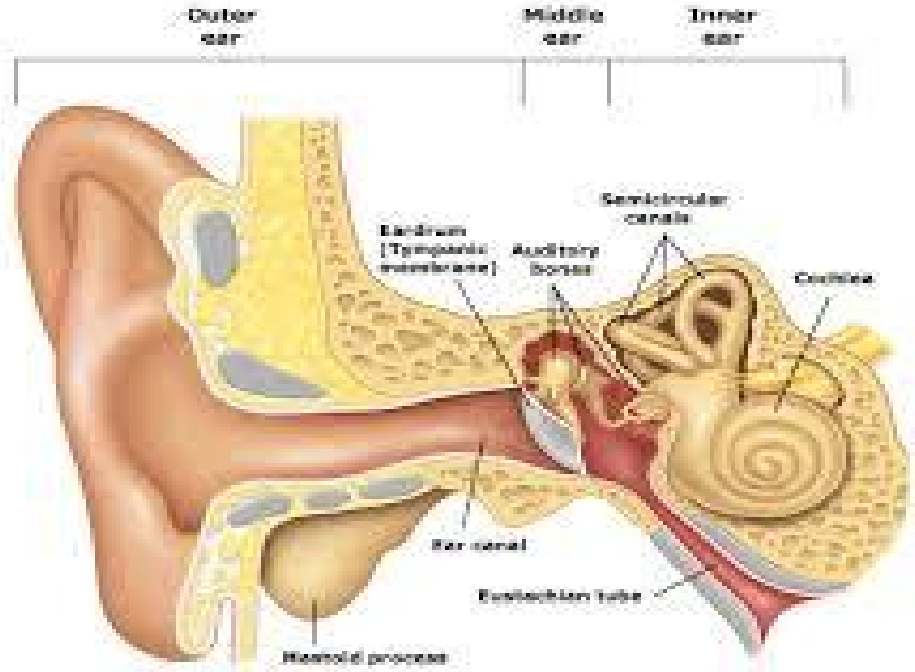
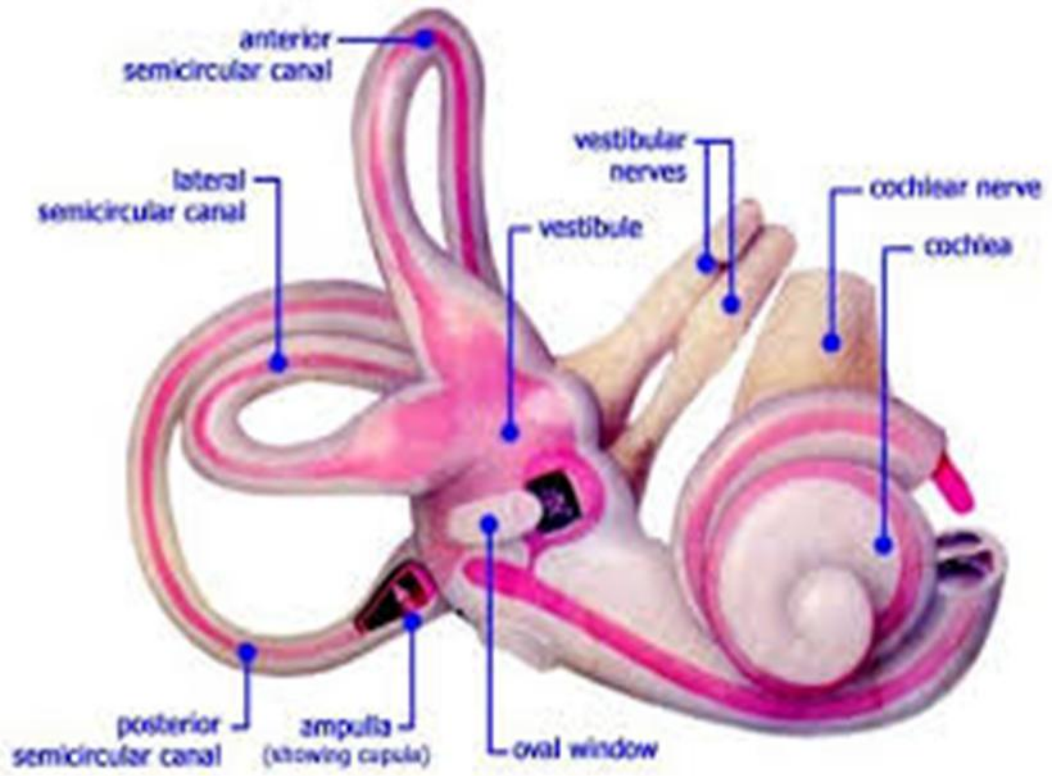


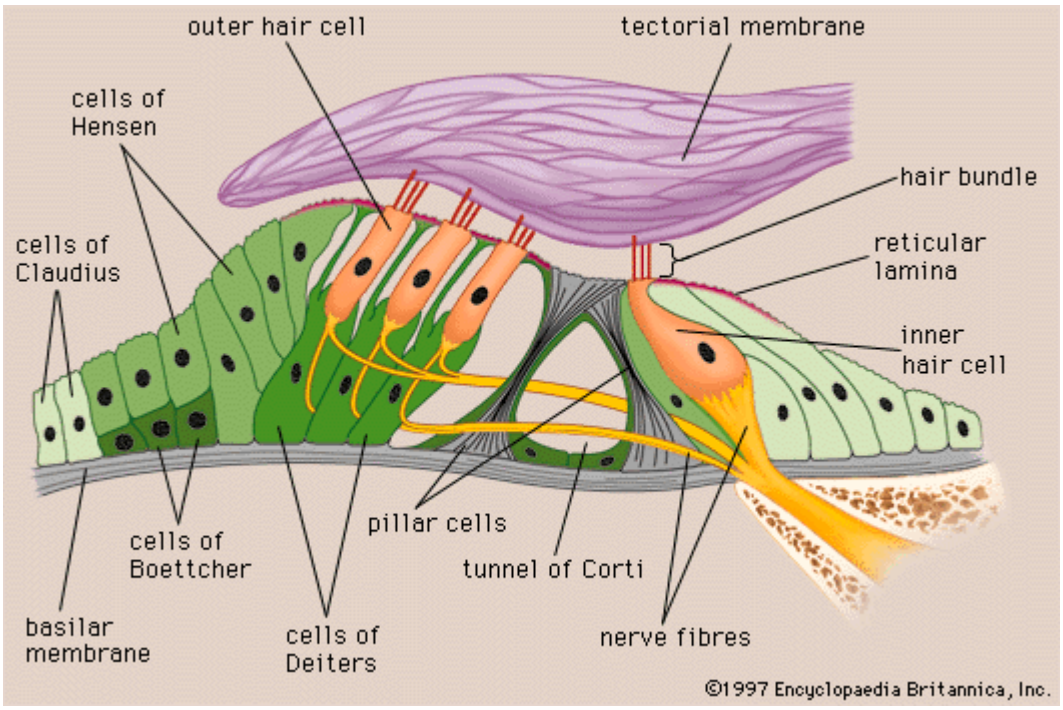
Fig :2 Schematic Diagram of Inner Ear



The ear is the organ of hearing and equilibrium[6].It is divided into three parts viz. The External ear, the Middle ear and the Internal ear. The internal ear houses two organs, viz., (i) Cochlea, meant for audition (hearing), and the (b) vestibular apparatus, meant for maintenance of equilibrium and posture. It is to be noted that the vestibular apparatus is not concerned with hearing. A partition wall, called the tympanic membrane (ear drum) separates the external ear cavity from the middle ear cavity.

MECHANISM OF HEARING

Fig :3 Schematic Diagram of Organ of corti



When the sound wave travels through the medium of air, it strikes the tympanic membrane[6] → the vibration of the tympanic membrane are transmitted through the middle ear ossicles, to reach the foot plate of stapes →the stapes now begins its rocking movement → perilymph of scala vestibule now moves →pressure changes in the perilymph is now transmitted through the Reissner's membrane and endolymph of scala media to the basilar membrane →the basilar membrane moves up and down →as a result ,the hair cells are pulled and move rather side ways. This side ways bending causes stimulation of the nerve fibers which emerge from these hair cells and as a result ,action potential,that is ,nerve

impulses, develop in the cochlear division of the VIIIth nerve; these impulses on reaching the appropriate part of the brain produces the sound perception of the subject. Two points in the above accounts need emphasis:

i. If frequency of the sound wave is high, the basilar membrane vibrates only at its basal side; whereas, in low frequency sound, the maximal vibration of basilar membrane is near its apical side.

ii. The emerging dendrons of the cochlear nerve, which are arising from the hair cells, arise from an area of the cell which is close to the origin of the hair. It is possible ,that a neurotransmitter bridges the gap between the base of the hair and the origin of the nerve.

AUDITORY PATHWAYS

1. The axons of the spiral ganglion that innervate the hair cells form the cochlear division of VIII th nerve[6].The 'auditory' nerve enters the medulla and ends in ventral and dorsal cochlear nuclei,thesite of the first synapse.

2. Second order neurons from the cochlear nuclei end in superior olive and trapezoid body on both sides of the brain stem from where third order neurons take origin.

3. Third order neurons pass up via a variety of pathways in the lateral lemniscus to the inferior colliculi of both sides.Some of these fibers also send collaterals to the reticular formation and medial geniculate bodies in the thalamus.

4. From the inferior colliculi many fibers project and relay in the medial geniculate body neurons finally project to the to the primary auditory cortex (area 41) which lies in the superior

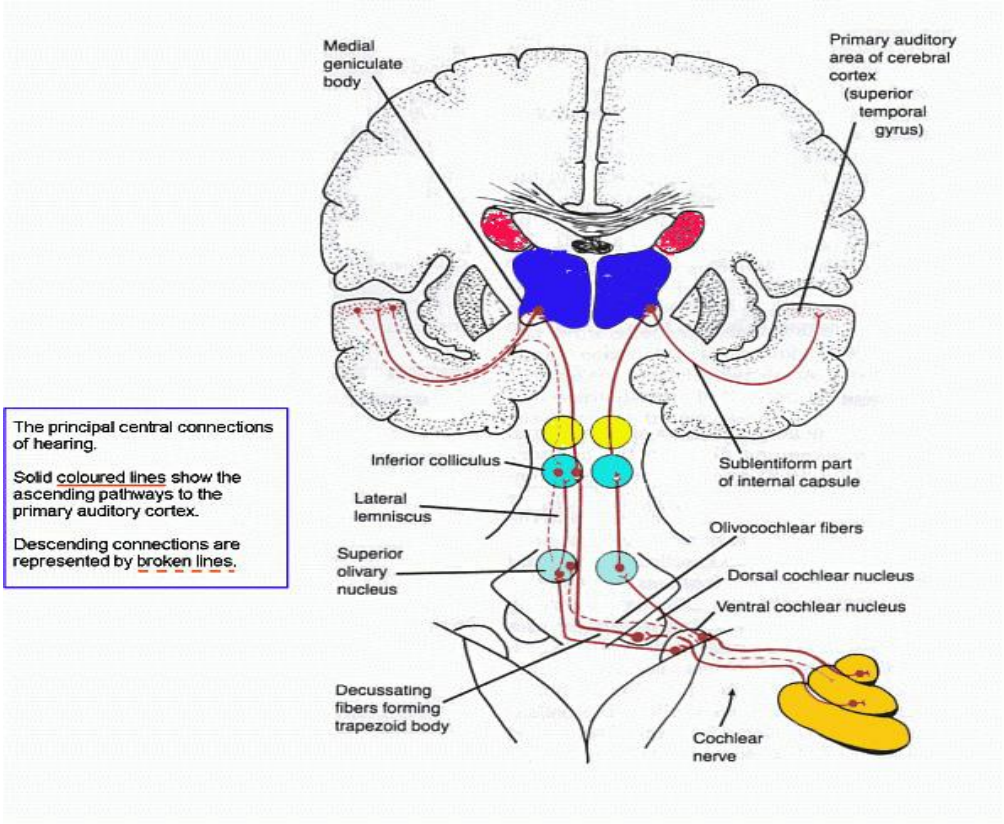
Portion of the temporal lobe located in the floor of the lateral cerebral sulcus. Here nerve impulses are perceived as sound i.e.it receives and perceives auditory informations such as **loudness ,pitch, source and direction of sounds.**

5. Auditory association areas[6] : area 22 ,21&20

(i) Area 22: **Wernicke's area**. It is located in superior temporal gyrus behind area 41, 42 in the categorical hemisphere i.e. dominant hemisphere .It is concerned with comprehension i.e. interpretation and understanding of auditory and visual informations.

(ii) Area 21 and 20 .These areas are located in the middle and inferior temporal gyrus and are concerned with interpretation and integration of auditory short-term memory without impairing visual memory.

Fig:4 Schematic Diagram of Auditory Pathway



BIOPHYSICS:

Normal nerve cell membranes have a stable resting and trans-membrane potential , because there is equal distribution of positively charged ions outside and negatively charged ions inside the cell membrane. The potential difference between two points is 0 inspite of a potential difference across the cell membrane[6].The action potential current occurs, due to the movement of Na+ and K+ ions which is responsible for flow along the cell membrane which is resisted by the intervening tissue and this resistance is known as **impedance.**

Voltage = current x impedance

Voltage represents the difference of voltage between two points .The action potential amplitude is shown in millivolts(mV) or microvolts(μv) ; current in milli-amperes (mA) ,and impedance in micro-ohms (mΩ).Time duration are in milliseconds (ms) or microseconds (μs). The action potential originating in nerves are displayed and expressed as wave form ,in which amplitude varies with time[6].

1. ELECTRODES :

(i) There are three types of electrodes : active ,reference and ground.The action potential is measured between active and reference electrodes whereas the ground electrodes serves as a ‘zero’ voltage reference point.

(ii) They are made up of platinum ,stainless steel, gold. Silver or gold electrodes has the advantage of stable electrode polarization potentials which results in noise free recording.

(iii) In clinical practice, two varieties of electrodes are used: surface and needle electrodes. In general, surface electrode is preferred, as using needle electrodes have a greater chance of infection.

(a) The surface electrodes are in the form of disc, cup or ring ,and are used in nerve conduction and motor evoked potential recording.

These electrodes are placed in position with the help of electrode paste or jelly, which is gently rubbed on the skin and then applied for proper contact.

(b) Needle electrode is commonly used for EMG study[6].They consist of Teflon coated stainless steel wire which tapers to a sharp tip.

2. AMPLIFIER:

A variable degree of amplification, upto 5x10 folds is needed before being displayed because of the following reasons:

(i) Biological signals are very small.

(ii) Intrinsic impedence of the electrode .It varies with frequency and electrode type used. The concentric needles have a higher impedence.

(iii) Impedence of electrode –skin.

3. FILTER :

It is a device that relatively allows a particular range of frequency from a signal. It is required for eliminating the noise and useful for bringing out the characteristics of the waveforms i.e.optimising the recording.

(i) The low frequency filters remove the slowly changing low frequency components and allows the higher frequencies to pass through. Therefore, it is also called as **high pass filter.**

(ii) The high frequency filters remove the rapidly changing high frequency components and allow the low frequency (upto 100 Hz) to pass through; therefore also called **low pass filters.**

4. AVERAGER :

(i) It extracts very small signals which are hidden or burried in large noise; for example:

(a) evoked potential are burried in EEG noise;and

(b) sensory nerve action potential in EMG noise.

(ii) By averaging, the time locked signals become prominent and are stored in the memory of the equipment ,while the noise which is occurring randomly is cancelled out. Alternatively ,the noise can be time locked and can be rejected subsequently.

5. DISPLAY:

Two methods of waveform display are in use:

(i) Analogue oscilloscope display

(ii) Computer based digital video display

i. Analogue oscilloscope display[6] : Here the action potential signals are directly displayed on cathode ray oscilloscope following amplification and filtering. It can redisplay the waveform but certain details of waveform may be lost.

ii. Computer based digital video display: Here an analogue to digital converter (ADC) and digital processing technique are used; therefore, the signals can be redisplayed with greater sensitivity without any loss of waveform accuracy.

6. STIMULATOR:

It is required for nerve conduction and evoked potential studies. Two types of stimulator are in use: Electrical and Magnetic.

(i) Electrical stimulators[6] : It is of two types:

(a) Constant current stimulator : It delivers a constant current to the subject over a wide range of stimulating electrode impedence. Thus it is more stable and useful in repetitive nerve stimulation and evoked potential studies.

(b) Constant voltage stimulator : It delivers a fixed voltage between anode and cathode.

(ii) Magnetic stimulators : They are used for non-invasive stimulation of motor cortex,spinal cord and peripheral nerves.

7. SENSITIVITY (or gain) SWEEP SPEED:

The latency and duration of an action potential are influenced by sensitivity (i.e.gain) and sweep speed.

(i) On high sensitivity ,there is shortening of latency due to visualisation of smaller deflection from the base line.

(ii) Increase in sweep speed results in shortening of latency.

8. SIGNAL TRIGGER:

It is useful for isolating and displaying the action potentials for their quantitative to the start of sweep.

9. DELAY TIME:

It continuously samples and stores into the memory ongoing action potential activities[6].The action potential when exceeds the triggering values, it is extracted from the memory and displayed.

EVOKED POTENTIAL :

Evoked potential which occurs in the group of neurons in reaction to stimulation of a sense organ which can be recorded by surface electrodes are known as Evoked potential[7].

BRAINSTEM AUDITORY EVOKED POTENTIAL:

Brainstem auditory evoked potential uses a click stimulus that produces a sequential activation of brainstem auditory pathway (ie) reaction from the cochlea, the signal that goes along the auditory pathway from the cochlear nuclear complex to the inferior colliculus in midbrain generates wave 1 to 5 ; occurring 10 milliseconds after auditory stimuli.

Brainstem Auditory Evoked Potentials / Auditory Brainstem Response (ABR).

The potentials recorded from the ear and vertex in response to a brief auditory stimulation are called Brainstem Auditory Evoked Potentials[7].These are produced within 10msec. by a brief click stimulus. A series of potentials are generated corresponding to sequential activation of peripheral, pontomedullary, pontine and midbrain portion of auditory pathway. It helps to assess:

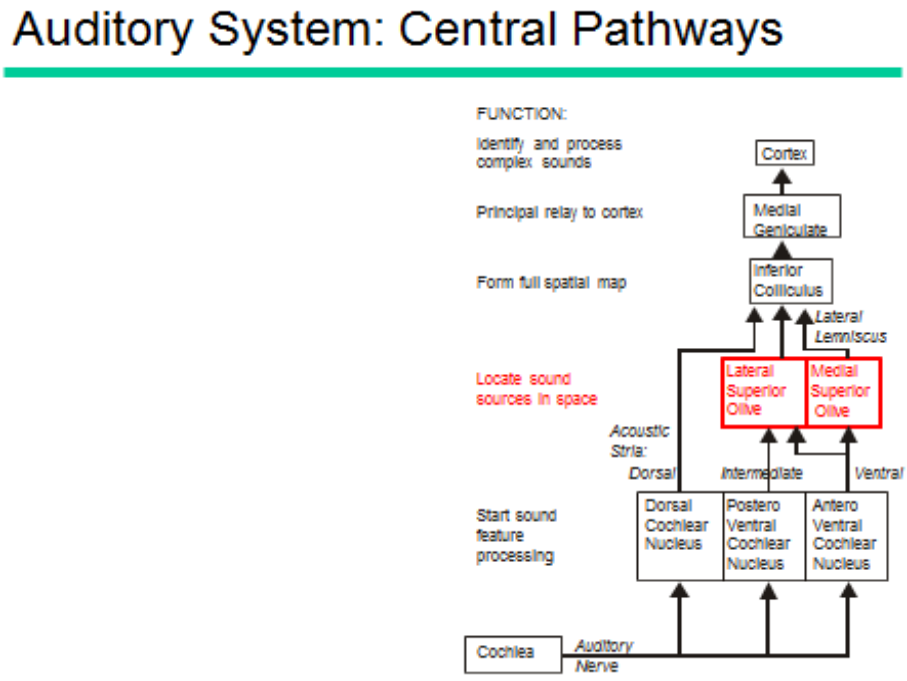
(i) Conduction through the auditory pathway upto midbrain.

(ii) Severity of hearing deficits; and

(iii) Middle portion of brainstem functions.

USES OF BAEP: Clinically BAEPs are used :

1. To assess hearing in uncooperative patients and very young children.
2. To detect degree of hearing loss in infants.
3. To assess the functions of the midpart of the brainstem.



The axons of the spiral ganglion that innervate hair cells of the ear form the cochlear nerve. The first order of neurons terminates in the cochlear nuclei in the medulla from where the second order of neurons arises and ends in the superior olivary nucleus. The third order of neurons originates from the superior olivary nucleus and ascends the lateral leminiscus to project onto the inferior colliculus which is the centre for auditory reflexes. From the inferior colliculi, many fibres project to the

medial geniculate body in the thalamus and from there to the primary auditory cortex (area41)[7].

Waves of BAEP

Five or more distinct waveforms are recorded within 10ms of the auditory stimulus. These waveforms are named wave I,II,III IV and V.If the recording continues ,a few more positive and negative waves are recorded.

Wave I :Orginates from the peripheral portion of the eighth cranial nerve adjacent to the cochlea.

Wave II :Orginates from the cochlear nucleus.

Wave III :Orginates from the superior olivary nucleus.

Wave IV : Orginates from the lateral lemniscus.

Wave V :Orginates from the inferior colliculi.

Factors that affect BAEP

1. Age The latency of BAEP is affected by age, especially in early childhood. Latency is age dependent up to two years. The effect of age is more pronounced in premature infants. Older adults have slightly longer I to IV interpeak latency compared to younger individuals[7].
2. Sex Women have shorter latency and higher amplitude of BAEPs.

3. Height The height of the subject has no direct correlation with latency or amplitude of BAEPs.
4. Temperature Increased body temperature decreases the latency and decreased temperature increases the latency of BAEP.
5. Drugs Barbiturates and alcohol prolong the latency of wave V. These drugs affect latency by decreasing the body temperature instead of directly acting on the auditory pathway.
6. Hearing loss Hearing deficit affects BAEPs. Therefore, hearing tests, especially to detect conductive deafness and examination of the ear to diagnose ear block by cerumin, should be done prior to recording BAEPs.

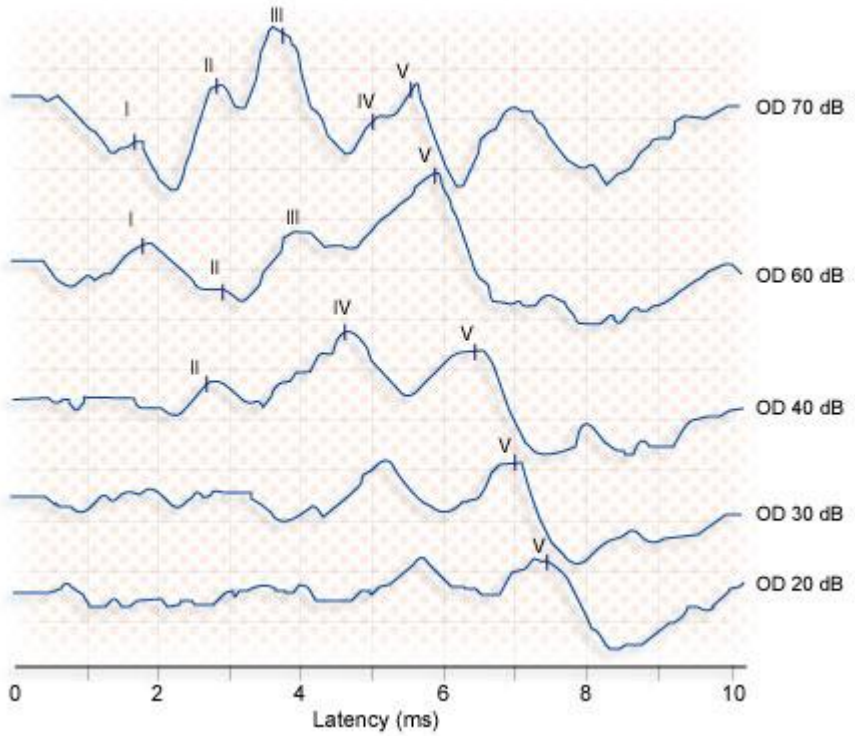


Fig : 5 Schematic Diagram of Wave forms

Wave I

Characteristics

1. This is the first prominent upgoing peak in the ipsilateral ear recording channel. It is reduced or absent from the contralateral ear recording channel.
2. It appears 1.4 ms after the stimulus.
3. The amplitude of this wave can be increased by using horizontal montage, external canal needle electrode, nasopharyngeal electrode, increasing stimulus intensity or decreasing the stimulus rate.

Clinical application As it originates from the eighth nerve this wave is preserved in patients who have only central problems. But, those who have peripheral hearing impairment have reduced or absent wave I. (wave II to V remain relatively normal).

Wave II

Characteristics

1. This is a poorly defined wave.
2. It appears as a small peak following wave I. It may appear in the downgoing slope of wave I or in the up going slope of wave III.
3. It is more prominent in the contralateral channel recording where it has a slightly prolonged latency compared to the ipsilateral recording.

Clinical application It is absent in lesions of the cochlear nucleus[7].

Wave III

Characteristics

1. This is a prominent up going peak.
2. It is smaller and appears earlier in the contralateral channel.
3. It may sometimes appear as a bifid wave (with two peaks).

Clinical application It is reduced or absent in lesions of the superior olivary nucleus.

Wave IV

Characteristics

1. This is a very small wave that usually appears in the up going slope of wave V.
2. Sometimes it may be absent or may appear as a very small wave at the peak of wave V giving it a bifid appearance.

Clinical application It is absent in lesions of the lateral lemniscus.

Wave V

Characteristics

1. This is the most prominent peak in BAEP.
2. It appears 5.5 ms after the stimulus.
3. It starts usually above the baseline immediately following wave IV.

Clinical application It disappears in diseases affecting the inferior colliculi.

Abnormal BAEPs: abnormalities include:

(i) Absence of wave I :It is seen in

(a) Large tumour damaging VIII nerve

(b) VIII nerve ischemia

(ii) Absence of waves beyond wave I :

Causes : (a) acoustic neuroma

(b) Meningioma

(c) Demyelinating disorders.

(iii) Absence of waves IV and V:

Causes: (a) multiple sclerosis

(b) hydrocephalous

(c) The right to left latency asymmetry exceeding 0.5msecis seen in acoustic neuroma.

Measurement of BAEP waveforms

The following parameters are measured for analysing the waveforms of BAEPs:

1. Absolute latency and amplitude
2. Interpeak latencies
3. Amplitude ratio of V/I
4. Inter – ear –interpeak difference.

Absolute latency and amplitude

The absolute amplitude is measured as the height from the peak of the wave to the trough of that wave. The absolute latency is measured as the distance (expressed in ms) from the beginning of the first wave to the peak of that wave.

Interpeak latencies

The interpeak latencies (IPLs) commonly measured are I-V, I-III and III –V. This is measured as the difference between the peak of both the waves(expressed in ms)[7].

I – V interpeak latency

1. The normal value is 4.5 ms.

2. It represents conduction from the proximal part of the eigth nerve through pons to the midbrain.

3. It is slightly less in females and more in elderly men.

4. It is prolonged in :

- Demyelination
- Degenerative diseases
- Hypoxic brain damage

I – III interpeak latency

1. The normal value is about2.5 ms.

2. It measures conduction from the eigth nerve across the subarachnoid space into the core of lower pons.

3. It is prolonged in:

- Inflammation or tumour of the eigth nerve
- Disease at the pontomedullary junction
- Guillain – Barre syndrome.

III – V interpeak latency

1. The normal value is about 2.4ms.

2. It measures conduction from the lower pons to the midbrain.

3. It is prolonged in prolongation of I –V IPL. The isolated prolongation III – V IPL is not considered significant.

Amplitude ratio of V/I

Wave I is generated outside and V is generated inside the CNS. Therefore, the V/I ratio compares the relationship of the signal amplitude.

Normal value The ratio is normally between 50 percent and 300 percent.

Clinical implication If the ratio is less than 50 percent, this suggests small wave V, which indicates a central impairment of hearing. If the ratio is more than 300 percent ,this suggests small amplitude of wave I, which indicates peripheral hearing impairment.

REVIEW OF LITERATURE

Neurophysiology of Evoked Potentials :

The first human brainstem auditory evoked potential was fully described by Jewett and Williston in 1971, and Sohmer and Feinmesser in 1967 attributed their origin in evoked potential.

The developments in clinical neurophysiology are related to invention of electricity. Lugigalvani in 1971 discovered that nerves were a good conductor of electricity.

Duchene in 1833 discovered the neuromuscular disease and developed the techniques for electrical stimulation.

Helmholtz measured the conduction velocity of nerve in frog by mechanically recording the muscle twitch in 1850.

Carlo Matteucci from Italy investigated the muscle nerve preparation and formulated the electrophysiological concept based functioning of the nervous system.

Du Bois Raymond in 1851, recorded the action potential of voluntarily contracting muscle using jars of liquid as eletrodes which was the beginning of electromyography.

Erb in 1861 was the first to find the method of electro diagnosis based on faradic and galvanic current. He was the one who demonstrated the increased electrical irritiblity of motor nerves in tetany, known as Erb's phenomenon.

In 1994, Williams GH[1], stated that in essential hypertension auditory evoked potential wave abnormalities are common due to central nervous system dysfunctions.

In 1996, O.P.Tandon, Daya Ram &R.Aswathi[2] stated that Brainstem auditory evoked potential responses were recorded from essential hypertensives and showed significant prolongation of absolute peak latencies of wave I,II,V and interpeak latency of wave III-V indicating involvement of brainstem auditory pathways.

In 1999, Chen Y L, Ding[3] Y P a case control study was carried out in Kenya with 50 elderly, hypertensive with ABR showed a relationship between hearing loss and arterial hypertension.

In 1990, Markova M.[8] Stated that a report was submitted after analyzation of 50 hypertensives with hearing impairement showed arterial hypertension is an important risk factor for hearing loss.

In 2004, Baraldi GS, Almeida LC,Borgea[9] ACLC observed that the systemic arterial hypertension and hearing loss have important prevalence in elderly population.

In 1989, Katz J[10]. Says that proper supply of oxygen and nutrients are needed to maintain alive of human body ,and maintain structure and functional integrity of heart and blood vessels.

In 1983,Nagahar K, Fisch U, Yagi N[11] stated that Hypertension ,the most common cause for structural change in heart and blood vessels

.

In 1996, Rarey KE, YL, Gerhardt KJ, Fregly MJ, Garg LC, Rybak LP.[12] Observed that arterial hypertension causes ionic changes in cell potentials causes hearing loss.

In 2004,Ferrerira DR[13], Silva AA,Kornet L, Hoeks APG, Janssen BJA, Major factor affects health today is chronic diseases caused by genetics, life style, environment and aging itself. for maintaining their physical and mental functions until close to death we shouldpay attention on their healthy life.

In 2001,Bachor E[14], selig YK, Jahnke K, Rettinger G, Kaemody Cs stated that progressive or sudden hearing loss is caused by high pressure in vascular system cause inner ear damage which is supplied by anterior inferior cerebellar artery supports the inner ear artery divided into cochlear artery and anterior vestibular artery.

In 1994, Ohinata Y[15], Makimoto K, Kawakami M, Takahashi H. Stated that the pathophysiology underlying hearing complaint in hypertension is Due to increased blood viscosity it reduces capillary blood flow and it reduces oxygen transport causing tissue hypoxia.

In 1998,Antikainen RL[16], Jousilahti, P,Tuomilehto J, Observed that methodology of study is focused on middle aged individual and excludes specific disease which impairs hearing.

In 1990, Carrasco VN[17], Prazma J, Faber JE.Gates GA, Cobb JL,D' Agostinho RB, Wolf PA 1993,Stated that the cause for sensorineural hearing loss with aging is due to microcirculatory insufficiency due to vascular occlution caused by emboli, haemorrhage or vasospasm because of microangiopathy caused by hypertension.

In 2003,Marchiori LLM[18], Gibrin PCD ,Collet L, Berger –Vachon C, Desreux V, Morogon A. Cochlear degeneration in hearing loss is due to environmental factors, may be subject, such as noise, inhalation of

toxic substances, metabolic and circulatory alterations, infections, genetic inheritance.

JNC – VII Hypertension[19] 2003; controls and hypertensive subjects systolic and diastolic BP were measured according to this Joint National Committee.

In 1996, Karamitos DG, Kounis NG, Zarvras GM, et[20] observed that 30 ischemic heart disease patients showed significant change in peak latency and inter peak latency, BAEPs become noninvasive assessment for essential hypertension and IHD.

In 1990, Sismanis[21]A, Callari RH, Slomka WS, etal.stated that pathophysiology of auditory evoked potential inn essential hypertension is stretching –compression of cochlear nerve in brainstem due to intracranial hypertension.

Julius S, Petrin J[22]. Autonomic nervous and behavioralfactors in hypertension. In Hypertension: Pathophysiology, Diagnosis and management. LaraghOHanaBrenner BM (Eds) Raven Press Ltd. NewYork 1990: PP2083-2090.

In 2003, Guidelines committee ,European Society of Hypertension[23] –European Society of Cardiology guidelines for the management of arterial hypertension. .

In 1986, Hashimotto et al,[24] In 1982,Mbller AR, Jannetta PJ [25] observed that lesion of tegmentum of lateral pontomedullary junction lead to changes in ipsilateral BAEP after wave II.

In 1975, star and achor[26]were the first to report the effects on the ABR of CNS pathology in the brainstem.

In 1994 , Marsh and Smith[27] has shown that raised blood pressure in pre –eclamptic women may affect vascular responses of the blood vessels in brain and cause ischemic delay in P1 latency of visual evoked potentials.

In 1994, Sethi A, and Vaney N,[28] in brainstem auditory evoked potential proved that there is decreased sensory conduction because of cold pressure response.

Fisher CM.[29] Cerebral miliary aneurisms in hypertension.

Am ,]Paihol 1972; 6G: 313.

In 1971 ,Frederic MW[30]. Stated that the basic mechanism for central nervous system dysfunction in hypertension due to arterial and arteriolar spasm of cerebral blood vessels, combined microinfarction, fibrinoid degeneration and brain oedema in severe cases.

In 1982, McGhee TB[31], observed that hypertension induced central nervous system dysfunctions in the form of sensory or motor deficits, presents with symptoms of occipital headache, dizziness, vertigo tinnitus reduced vision.

In 1994, Pavfilov V V,Reid JL[32].stated that essential hypertension leads to dysfunction in mechanism of brainstem so it leads to sensory deficits due to interaction of sensory neuronal substrate.

Ziegler DK[33]. Hypertensive vascular disease in the brain.In Hand book of clinical neurology Vinken P,J, Bruyn GW (Ed) Elsevier Publishing Company New York 1972;12 : 552.

In 1981 OH SJ, Kuba T, Soyer A, Choi IS, Bonikowski FP, Vitek J[34] Stated that brainstem auditory evoked potentials are lateralisation of brainstem lesions.

In 1979, Nago S, Roccaforte P, Moody RA[35] ,observed that in experimental transtentorial herniation presents with unilateral pupillary dilatation and auditory evoked response showed changes in wave V.

In 1980, Tsubokawa T, Nishimoto H, Yamamoto T,et al[36]stated that to assess prognosis of comatose patients with brainstem auditory evoked potential is used for number of cases.

In 1982, Karnaze DS, Marshall LF,McCarthy CS, et al [37] stated that after closed head injury in coma for localisation and prognostic value monitored by auditory evoked response.

In 1975, Starr A, Achor LJ[38]: observed that reversible brainstem dysfunction and non reversible brain dysfunction has been differentiated by brainstem auditory evoked response.

In 1982, Hall JW, Huang –Fu M, Gennarelli TA[39]: stated that auditory function in acute severe head injury is monitored with auditory evoked response waves.

In 1985, Scherg M, Cramon D von[40] .observed that the wave generators of BAEP located in tegmentum of upper medulla and pons.BAEPs are very sensitive for detection of pontine haemorrhage.

In 1986, Stockard JJ, In 1977 Rossitter (Stockard JE, Sharbrough FW, J.J., J.F. Hughes[41],[42,43], stated that brainstem auditory evoked potential gives the information Of methodology, interpretation, and clinical application .

In 1985, Hammond EJ, Wilder BJ, Goodman IJ, Hunter SB[44], the study of BAEP and pontine haemorrhage have prognostic value and correlation of evoked potential findings with clinical signs and localization of lesion.

In 1980,Maurer et al[45],and Stocked et al 1986,observed that generators of wave limited by rarefaction and condensation clicks it may provide different value from same patient.

In 1981, Hashimoto I, Ishiyama Y, Yoshimoto T, Nemoto S[46] ,observed that haemorrhage in right tegmental pontine region BAEP showed loss of wave V after contralateral ear stimulation.

In1980, Hashimoto, I.,Y. Ishiyama, G.Totsuka& H.Mizutani[47].stated that more specific test for monitoring auditory function is intraoperative BAEP useful in microvascular decompression of CN V and CN VII.

In 1983, Chiappa[48], stated that lateralisation of BAEPs seen in unilateral bleeding in the pontinetegmentum on ipsilateral side. In demyelinating disease delayed latency of peaks and reduction of amplitudes and loss of waves are seen.

In 1996, Sabbatini M, Vega JA , Amenta F[49]. Stated that animal studies also consistent with development of neuropathy with hypertension, there is luminal narrowing leads to reduced vascular supply in hypertensive rats.

In 2001, Sabbatini M, Bellagamba G, Vega JA[50], et al observed that peripheral neuropathy in hypertensive rats are improved with antihypertensive treatment.

In 2004,Tomassoni D, Traini E, Vitaioli L, et al[51] observed that hypertensive rats shows peripheral neuropathy and decreased nerve conduction velocity and reduced percentage of class I fibers and increased percentage of class II & IV fibers in sciatic nerve of spontaneously hypertensive rats.

In 1983,Kanaya H, Saiki I, Ohuchi T, KamataK, Endo H, Mizukami M, Kagawa M,Kaneko M[52] Intracerebral haemorrhage in essential hypertension involves putamen, and extends into internal capsule it produce abnormal waves in BERA.

In 2001, Wang J, Liu YS, Liu SM[53]. Observed that significant prolongation in wave IV, V and VII presents with ABR also seen in rabbits with intra cranial hypertension.

In 1992, Picton TW, Taylor MJ[54], abnormality in BAEPs depends metabolism and myelination of CNS, so hypertension metabolic alteration in these produce abnormal latencies and inter peak latencies.

In 1986, Cant et al [55] stated that auditory pathway destruction leads to loss of BAEP waves not only due to compression of brainstem and also more likely a lesion of whole brainstem at the level of generator of altered wave, including structures lying rostral to this level.

In 1974 ,Hecox and Galambo[56] showed that the ABR could be used for threshold estimation in adults and infants.

In 1998, Souza LCA, PizaMRT, Machado CSS, Barcelos WCO, Britto DBO[57], Stated that BAEP is the instrument for assessing brainstem function in coma to predict the prognosis and diagnosis.

AIM AND OBJECTIVE

AIM :

To evaluate Brainstem Auditory Response in Essential Hypertension.

Objective :

To use Brainstem auditory evoked potential as tool to measure the subclinical abnormalities in patients with Essential Hypertension.

To assess the latencies and interpeak latencies in patients with Essential Hypertension.

MATERIALS AND METHODS

This study was conducted at the Institute of Physiology, Kilpauk Medical College, Chennai.

Study Design: Case control study.

Subject selection : Total sample size -80 , Essential hypertensive patients -40 ; controls – 40.

INCLUSION CRITERIA :

* Patients with Essential hypertension diagnosed by the department of medicine, Kilpauk Medical College.

  Male &Female between 40 – 60 years.

* Written informed consent.

Exclusion Criteria :

- Ischemic heart disease.
- Diabetes mellitus.
- Cerebro vascular disease.
- Alcoholism.
- Any neurological diseases.
- Renal disease.

- Hearing impairment.
- Drugs causing ototoxicity.
- Vitamin deficiency.
- Drugs causing neuropathy.
- Thyroid disorders.
- H/O Antiepileptics.

CONTROL - Healthy volunteers who are age & sex matched will be choosen as controls.

Screening procedures :

- Patients who qualify under the inclusion criteria will be enrolled in the study.
- Heart rate.
- Blood pressure .
- Height.
- Weight .
- Brief history to rule out ,diabetes ,IHD ,alcoholism ,drug intake.
- General clinical examination

EQUIPMENT DETAILS :

Brainstem auditory response studies will be carried out on a computerised Nerve conduction testing equipment**: Medicaid, computerised physiolab, Neuroperfect plus**

INSTRUMENT - MEDICAID – NEUROPERFECT PLUS

Ethical Considerations :

Institutional Ethical committee approval was obtained from Kilpauk Medical College Chennai – 10.

Subjects : The subjects were made to relax themselves before the test.The procedure and objective for this study was explained in their own language.A detailed clinical history about hypertension was collected from them. The written and informed consent was obtained prior to the individual were subjected to the study.The complete

examination of the ear was done for both the ears. Blood pressure measured for both control and subjects. The basic parameters of the subjects like height ,weight ,and pulsewas recorded. Brainstem auditory evoked potential were recorded in 40 hypertensive patients and 40 control of both the genders. Toestablish the reliability of the method, severalrepititions of auditory evoked potential recordings were done.

Precautions :

1. The subject should be properly instructed and motivated to provide full cooperation.

2. The room should be quiet and comfortable.

3. The skin of the scalp and mastoid should be grease free.

PREREQUISITES FOR THIS STUDY :

- The subjects were adviced to have a head bath with shampoo prior to the test.

- The individuals are adviced not to apply oil or hair spray on their head.

- The research room was made comfortable to the subjects ,calm and uniform temperature maintained.

- Subjects were instructed to sit on the wooden chair and keep their leg on the wooden block.

- They were instructed to remove their ornaments from the ear, neck and hand.

PROCEDURE: The recording of BAEP was done in Neurophysiology research laboratory, Department of physiology , Kilpuk Medical College using Neuropefect Plus – Medicaid Physiolab. The left and right ears were tested separately in all subjects.

RECORDING OF BAEP – INSTRUMENT SETTING

Requirements :

1. Recording electrodes

2. Amplifier and average

3. Electrode paste

4. Earphone.

Equipment set up:

Suggested Montage :

1. Channel : Ai – Cz - Active electrodes

2. Channel : Ac – Cz – Ipsillateral ear (Ai), Contralateral ear (Ac) or mastoid process.

3. Ground : Fz - Reference electrode ; Cz i.e.at vertex (it is regarded as a suitable location as waves II-V have a good amplitude at the vertex)

Recording conditions:

1. Filter

   a. Low filter cut :10 -100 Hz.

   b. High filter cut :3000Hz.

2. Amplification between 2,00,000 – 5,00,000

3. Sweep speed :1 msec/division.

4. Number of epochs :at least 100.

5. Electrodeimpedence should be kept below 5 kilo-ohms.

Stimulation options:

1. Sensitivity : 0.3 uv /division

2. 60db sensory level i.e.point at which the individual can barely appreciate the stimulus.

B.Steps :

1. Keep the instrument out of view of the subject.

2. Allow the subject to sit comfortably on a chair in a fully relaxed state.

3. The skin at the point of placement of electrodes is cleaned with ether or spirit.

4. Using electrodes paste or conducting jelly ,the recording (active) electrode are placed on both the ears, ipsilateral (Ai) and contralateral ear(Ac) or mastoid process as per 10 – 20 international system of EEG electrode placement ; the reference electrode is placed at the vertex i.e.at Cz ; the ground electrode is placed at Fz

5. Check the equipment set up as per technical recommendation of BAEP study.

6. The electrodes are connected through the preamplifier to the cathode-ray oscilloscope (C.R.O)

7. Give a brief click stimulus which is usually a square wave pulse of 0.1 msecduration.A click rate of 11-31 Hz are most commonly used in clinical practice.

8. Observe the effect of click intensity on BAEP .Analyse the BAEPs wave form and compare with the normal.

ElectroPhysiological Study:

Brainstem auditory evoked potential (BAEPs) constitute an objective hearing test[6]. These are the potentials recorded from the ear and the scalp in response to a brief auditory stimulation .The evoked potentials that appear following transduction of the acoustic stimulus by the ear cells ,create an electrical signal that is carried through the auditory pathway to the brainstem and from there to the cerebral cortex. When the signal travels, it generates action potential in all the fibres. These action potentials can be recorded at several points along the auditory pathway and even from the surface of thebody. BAEP assess conduction of the impulse through the auditory pathway up to the midbrain[6].

PARAMETERS STUDIED : The wave latency I ,II,III,IV, V and inter peak latency I-III,I-V, and III-V of waves were measured. The wave pattern in recording BAEP is 5 or more peaks within 10ms of stimulus. Initial 5 peaks have clinical value.

TABLE – 2

Anthropometric Measurements of cases (Hypertensives) with controls

Variables	Cases n=40 Mean ± SD	Controls n=40 Mean ± SD	P value
Age (yrs)	52.02 ± 5.34	51.74 ± 4.61	.80
Height (cm)	159.45 ± 2.69	158.0 ± 3.260	.34
Weight (kgs)	62.50 ± 3.58	59.33± 2.08	.00
BMI (Kg/m2)	24.59 ± 1.56	23.78 ± .91	.006

BMI – Body Mass Index

The parameters were analyzed using student independent ‘t’ test.

$P < 0.05$ is taken as significant.

Fig :3

Anthropometric Measurements of cases (Hypertensives) with controls

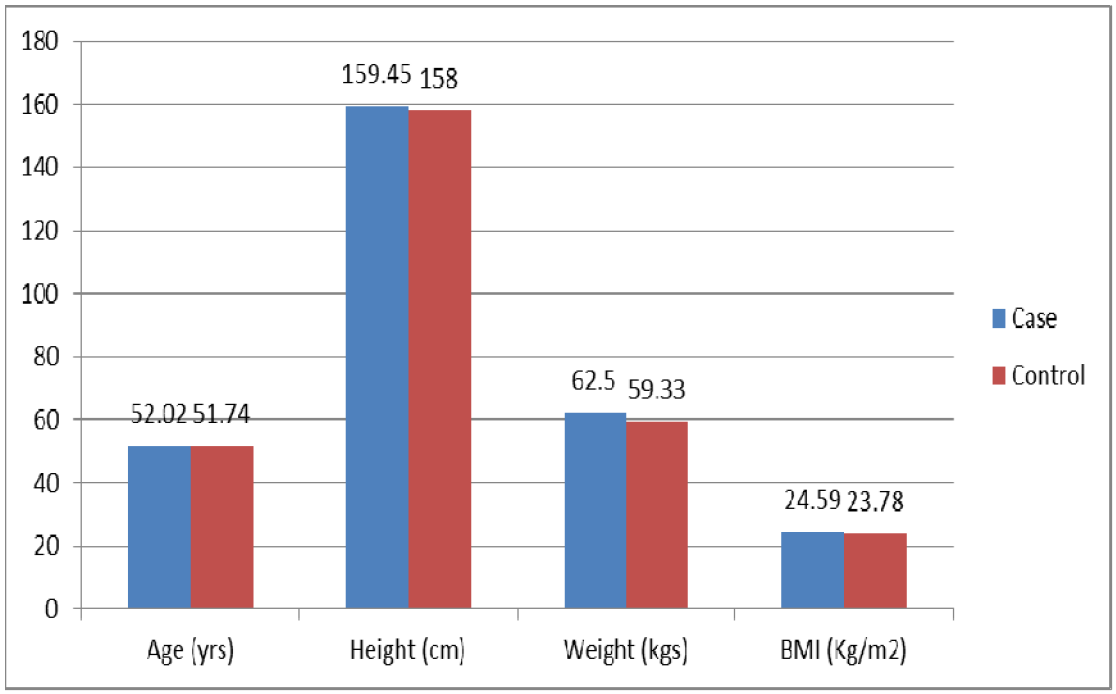


TABLE - 3

COMPARISON OF BLOOD PRESSURE IN CASE AND CONTROL

	CASE	CONTROL	P value
SYSTOLIC BP	159.6 ± 14.4	101.4 ± 8.31	.000
DIASTOLIC BP	122.8 ± 6.40	85.2±5.57	.000

The parameters were analysed using student independent ‘t’ test.

P <0.05 is taken as significant.

Fig:4

COMPARISON OF BLOOD PRESSURE IN HYPERTENSIVE SUBJECT AND CONTROL

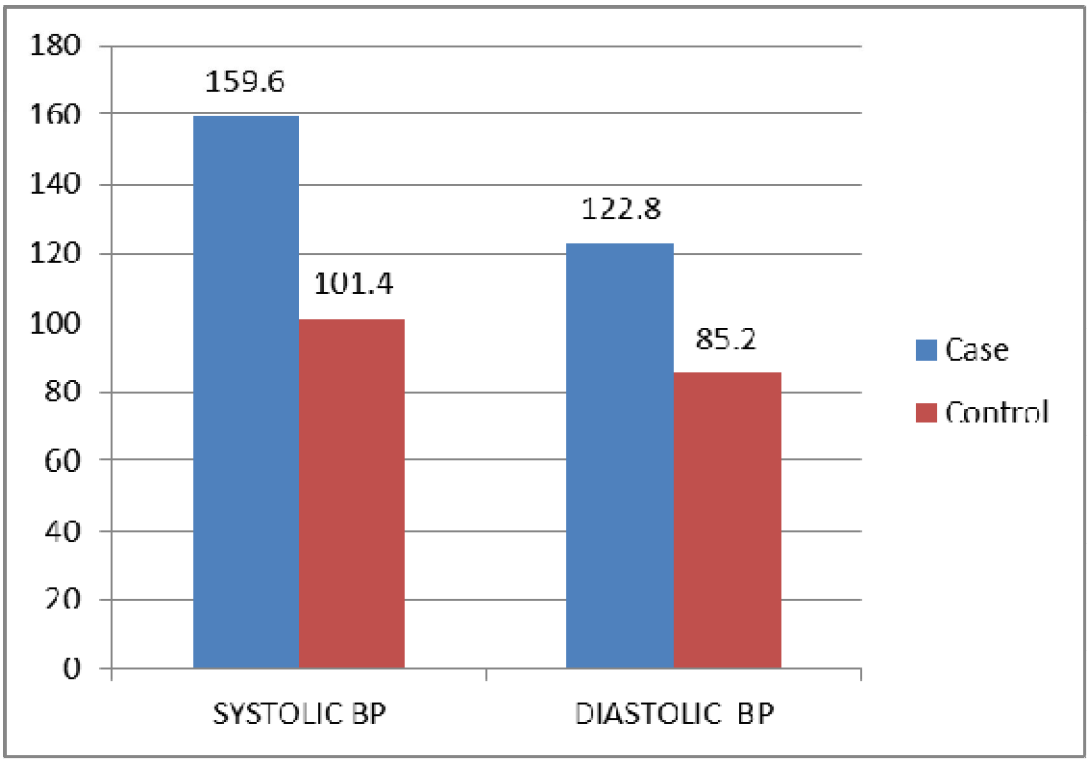


TABLE – 4

FREQUENCY DISTRIBUTION OF HYPERTENSIVE CASES

Category	No of Patients	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Stage I	20	140 – 159	90 – 99
Stage II	12	160 – 179	100 – 109
Stage III	8	180 – 209	110 – 119
Stage IV	0	>210	>120

TABLE -5

COMPARISON OF ABSOLUTE PEAK LATENCIES BETWEEN HYPERTENSIVE SUBJECTS AND CONTROL OF RIGHT EAR

Latencies (ms)	Case n=40 Mean±SD	Control n=40 Mean±SD	t value	P value
I	1.48±.59	1.25±.177	2.403	.019
II	2.05±.46	1.83±.40	2.213	.030
III	1.97±.46	1.736±.401	2.498	.015
IV	2.55±.581	2.107±.284	4.28	.000**
V	3.636±.98	2.742±.533	4.287	.000**

P< 0.05 is taken as significant.

P<0.01 is taken as highly significant.

Fig :5

COMPARISON OF ABSOLUTE PEAK LATENCIES BETWEEN HYPERTENSIVE SUBJECTS AND CONTROL OF RIGHT EAR

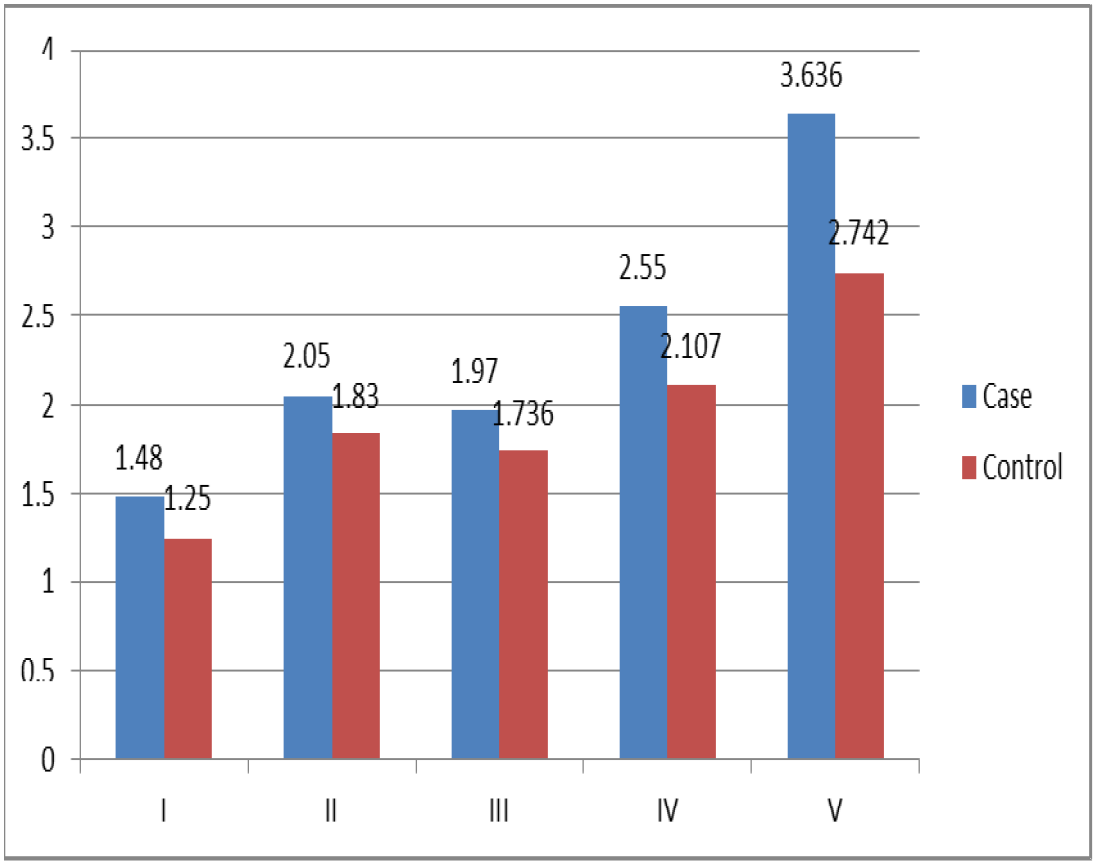


TABLE-6

COMPARISON OF INTERPEAK LATENCIES BETWEEN HYPERTENSIVE SUBJECTS AND CONTROL OF RIGHT EAR

Inter peak latencies	Case N=40 Mean±SD	Control N=40 Mean±SD	t value	P value
I – III	4.03±1.19	3.03±.548	4.767	.000**
III – V	1.19±.480	.95±.460	2.269	.26
I-V	1.48±.69	.92±.399	4.388	.000**

P<0.05 is taken as significant.

P < 0.01 is taken as highly significant.

Fig :6

COMPARISON OF INTERPEAK LATENCIES BETWEEN HYPERTENSIVE SUBJECTS AND CONTROL OF RIGHT EAR

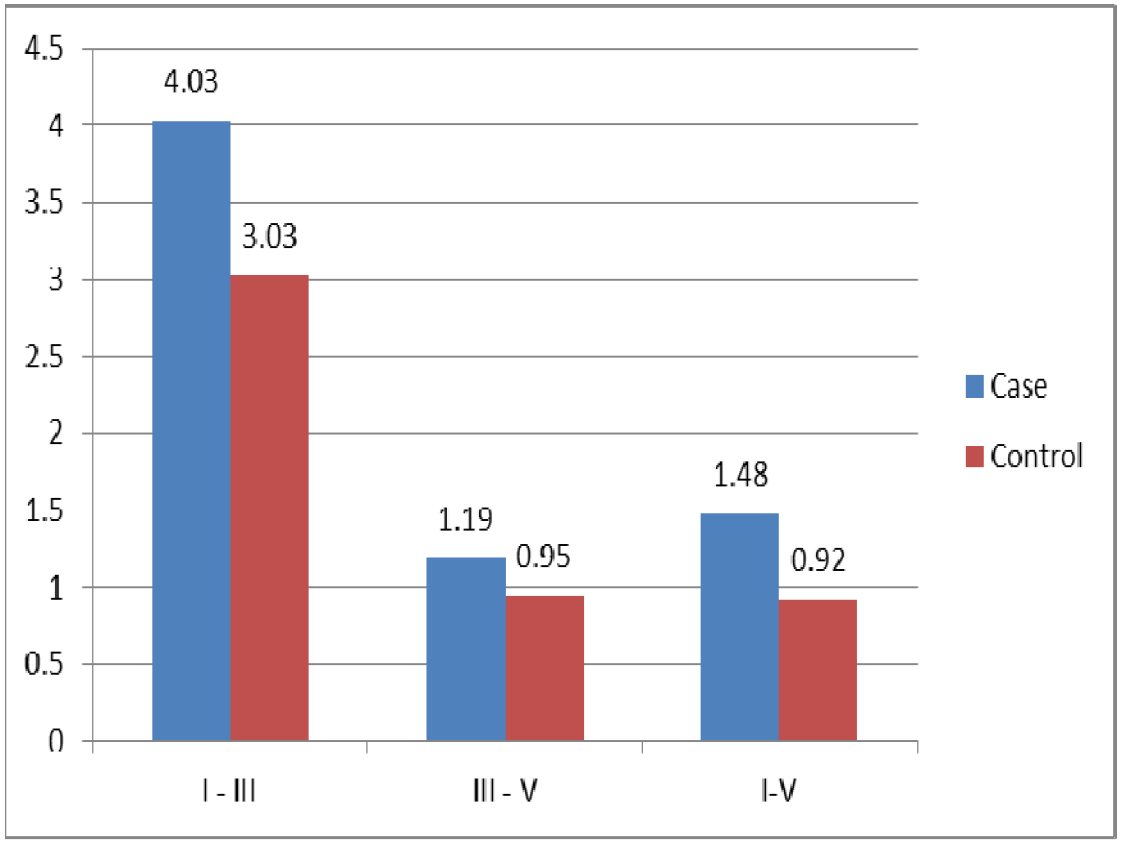


TABLE -7

COMPARISON OF ABSOLUTE PEAK LATENCIES BETWEEN HYPERTENSIVE SUBJECTS AND CONTROL OF LEFT EAR

Latencies(ms)	Case N =40 Mean±SD	Control N =40 Mean±SD	t test	P value
I	1.48±.460	1.205±.188	2.674	.009
II	2.046±.459	1.832±.398	2.213	.030
III	2.630±.538	2.20±.356	4.104	.060
IV	3.669±.985	2.935±.65	3.895	.070
V	4.121±1.11	3.284±.674	4.255	.000*

P<0.05 is taken as significant.

P < 0.01 is taken as highly significant.

Fig : 8

COMPARISON OF ABSOLUTE PEAK LATENCIES BETWEEN HYPERTENSIVE SUBJECTS AND CONTROL OF LEFT EAR

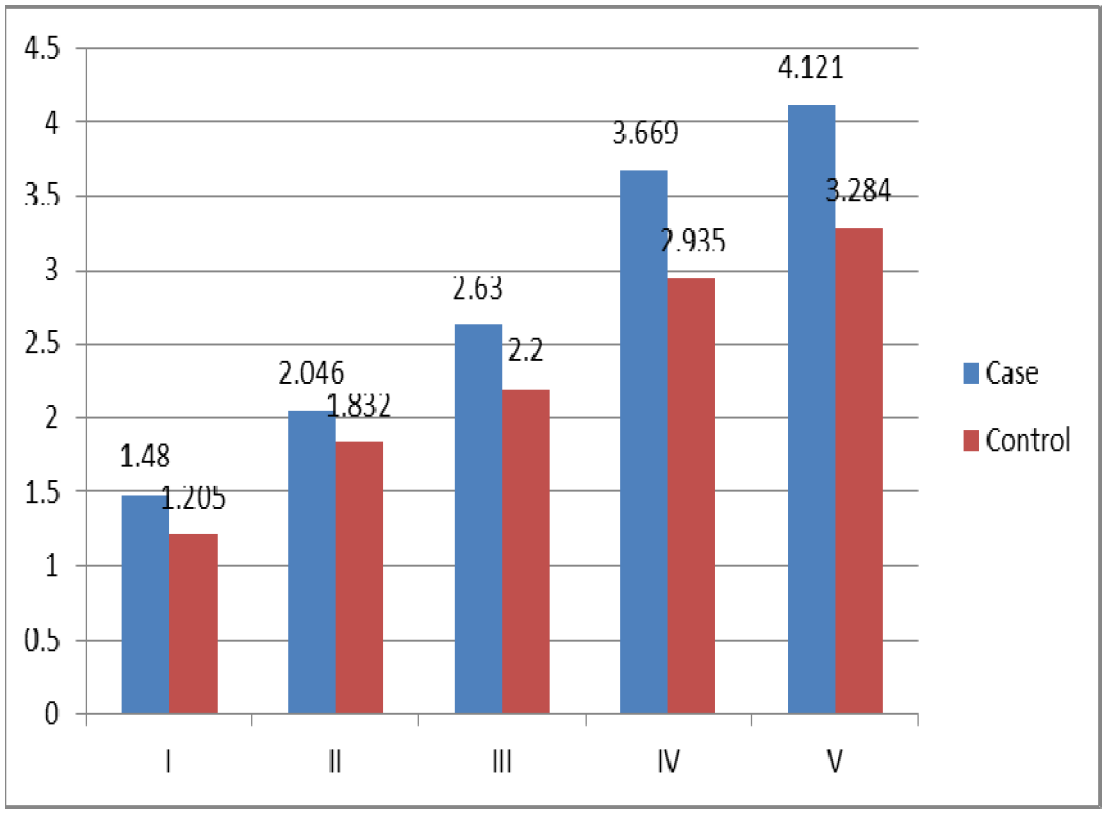


TABLE - 8

COMPARISON OF INTERPEAK LATENCIES BETWEEN HYPERTENSIVE SUBJECTSAND CONTROL OF LEFT EAR

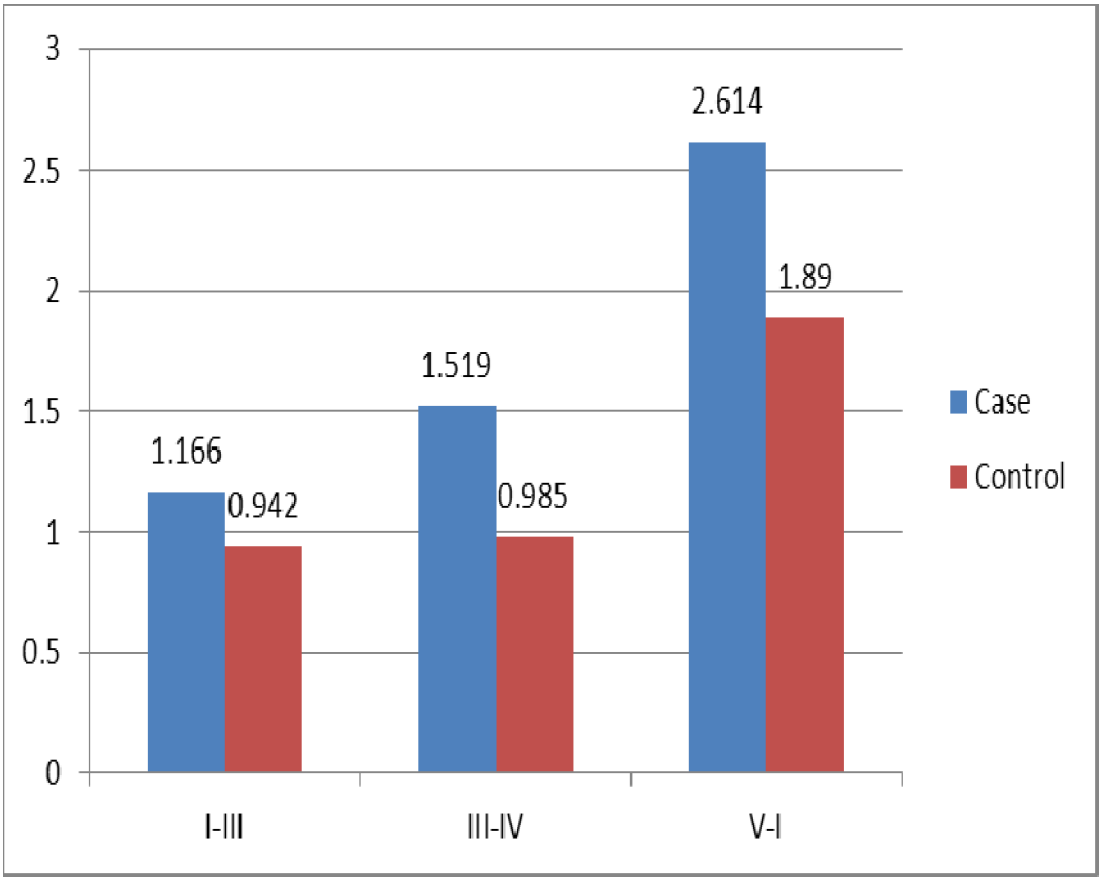
Inter peak latency	Case	Control	t value	P value
I-III	1.166±.404	.942±.38	2.504	.014
III-V	1.519±.730	.985±.474	3.838	.000**
V-I	2.614±.823	1.89±.698	4.163	.000**

P<0.05 is taken as significant value.

P < 0.01 is taken as highly significant.

Fig : 7

COMPARISON OF INTERPEAK LATENCIES BETWEEN CASE AND CONTROL OF LEFT EAR

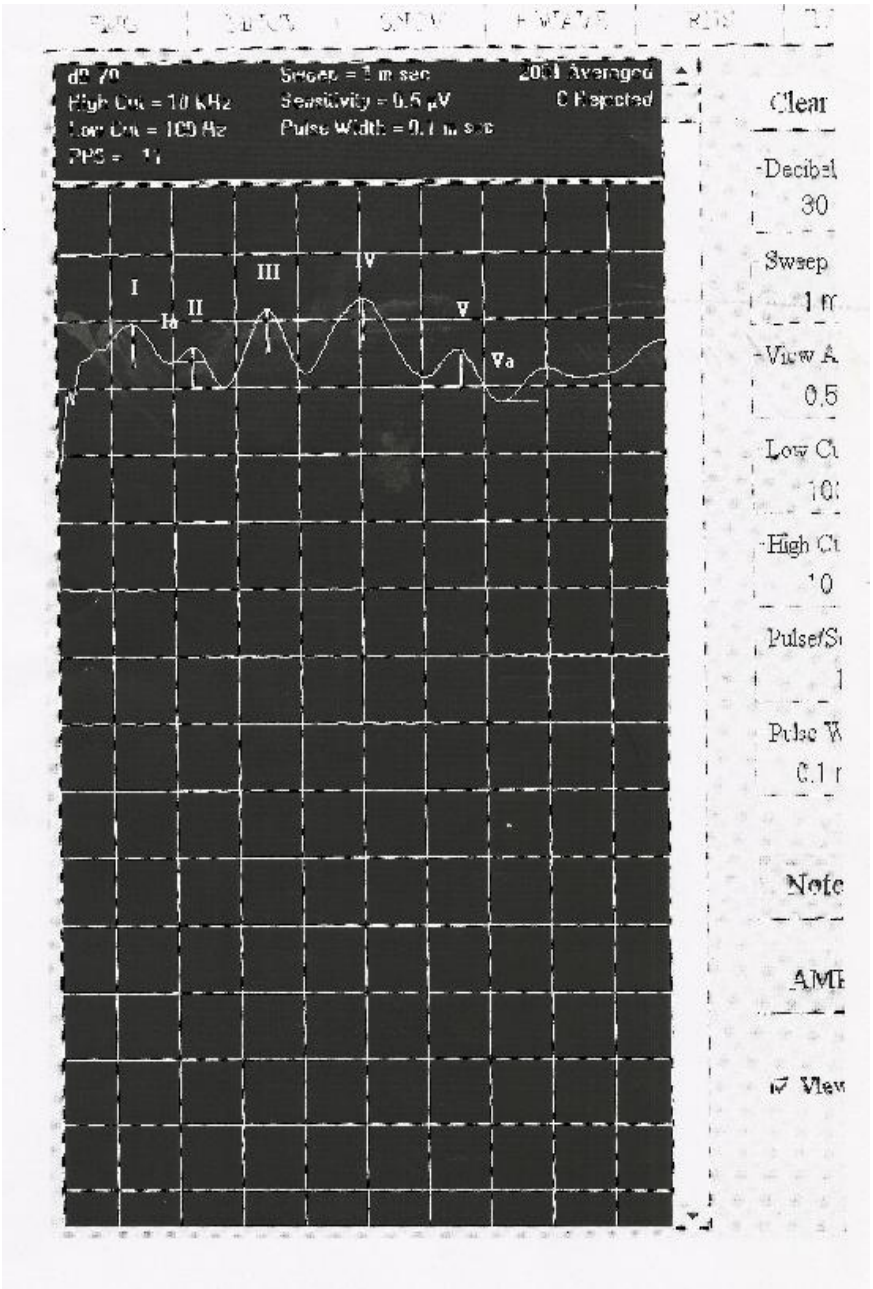


BRAINSTEM AUDITORY EVOKED POTENTIAL

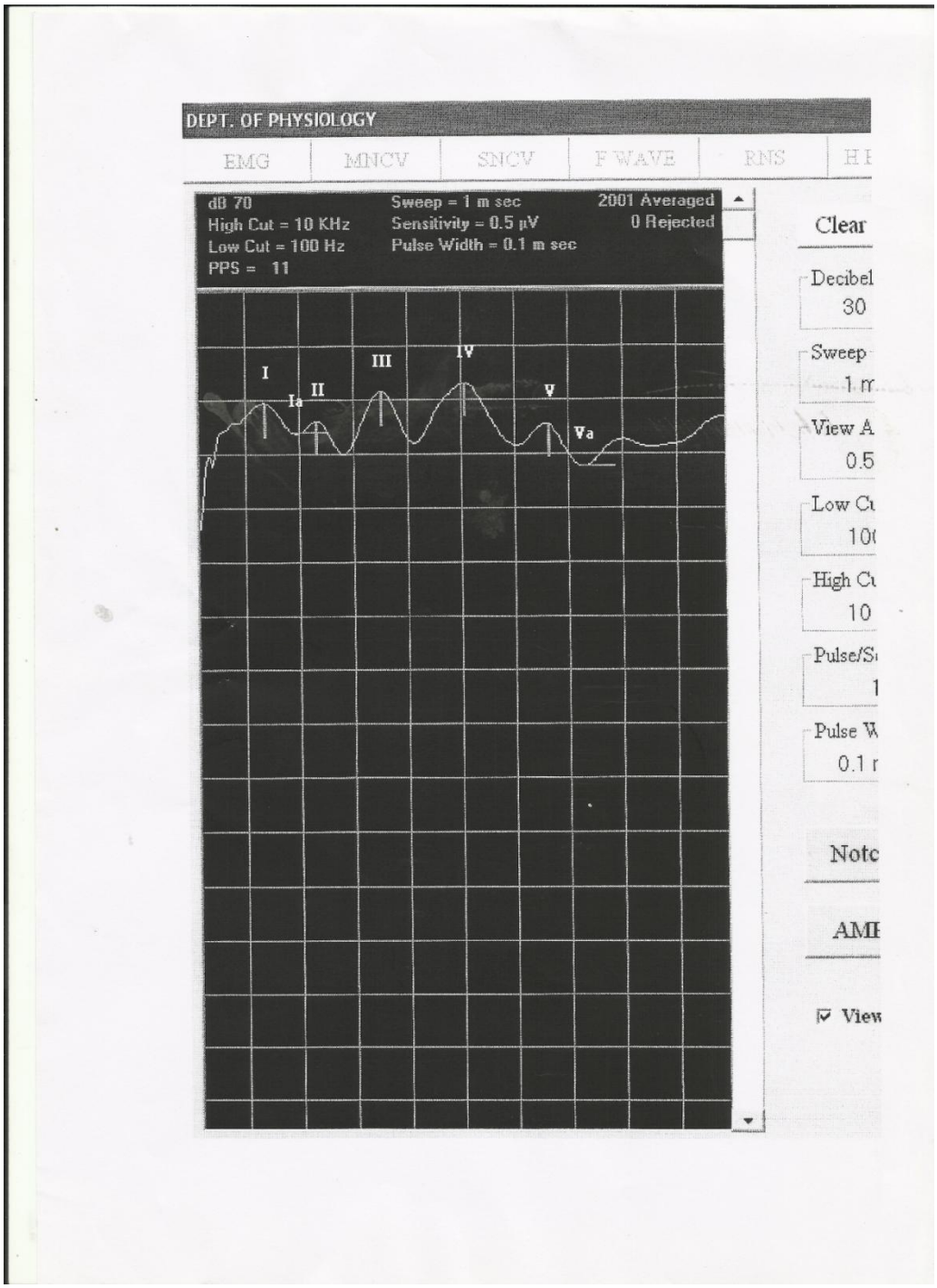
RECORDING OF A SUBJECT

BRAINSTEM AUDITORY EVOKED POTENTIAL RECORDING OF A SUBJECT

RECORDING OF BAEP WAVE FORMS IN RIGHT EAR



RECORDING OF BAEP WAVE FORMS IN LEFT EAR



RESULT

Control Group : There were 40 cases in this control group average age 51.74 ± 4.61 yrs, average weight of 59.33 ± 2.08, average height in cm 158.0 ± 3.260 ,systolic BP 101.4 ± 8.31, Diastolic BP 85.2 ± 5.57.Since the values of BAEPs of Right and Left ears did not vary significantly, average of two ears was calculated and these absolute peak latencies and inter peak latencies are given in table 5 ,6 ,7 & 8.

Hypertensive Group : There are 40 Primary Hypertensive patients ranging from 40 – 60 years of age with an average age of 52.02 ± 5.34 yr, average weight 62.50 ± 3.58 Kg, Height 159 .45 ± 2.69 cm ,Systolic BP 159 .6 ± 14.4 mmHg, Diastolic BP 122.8 ± 6.40 mmHg.

Hypertensive subjects were graded on the basis of raised BP ,as per criteria laid down by seventh report of the joint national committee on selection, evaluation and treatment of high blood pressure[15] .The average value of BAEPs two ears of absolute peak latencies are given in table 5,7interpeak latencies of both ears are given in table 6 & 8.

The female subjects both in control and hypertensive group had slightly lower values of BAEPs as compared to males but as these were not statistically significant. Composite data in each group were given in the tables.

In hypertensive group comprising of 40 cases ,8 cases belongs to stage III hypertensives showed abnormal BAEPs . All other cases of stage I (n=20), stage II (n=12) did not show significant change in latencies and amplitudes of waves when compared with controls.(Table 5,6,7,8)

Significant increase in absolute peak latencies in Right Ear on wave I ,III IV &V & inter peak latencies of wave (I – III) & (I – V).

Left Ear – increased absolute peak latency of wave I, &V & inter peak latencies on (III – V)& (V –I).

DISCUSSION

The aim of our study was to evaluate the effect of hypertension on ABRs, 40 patients with primary hypertension were selected between ages of 40 – 60 yrs ,along with 40 age and sex matched normotensives. In our study auditory threshold increased significantly.

In a study done by O.P.Tandon[2] et al, auditory evoked potential in stage – III essential hypertension showed, significantly prolonged absolute peak latency in wave I, II &V and Prolonged inter peak latency in wave (III -V)

Another study by Shilpakhullar, Navingupta and Rashmi Babbar et al, Auditory brainstem responses and Nerve conduction velocity in Essential Hypertension showed significantly increased absolute peak latencies of waves I, II & V compared to controls and increased IPL in wave (III –V). There is also another study that showed increased latencies of wave V, inter peak latency (V-I) and wave (III-V) were prolonged compared with normal elderly subjects.

Karamitos[20] et al, studied ABRs and measured the parameters of APL waves I to V and IPL (I-III), (III-V) & (V-I), and he found measured absolute peak latencies and interpeak latencies were significantly increased. Hence BAEPs become part of noninvasive assessment for IHD and Essential hypertensive subjects.[20]

There is another study wang J[53] et al showed significant prolongation of waves IV,V & VII of ABRs, were seen in rabbits with intracranial hypertension compared with controls. These abnormalities due to stretching –compression of cochlear nerve in brainstem, because of intracranial hypertension or primary edema.[17]

Similar correlation of Increased in P1 latency of VEP with increased BP in preeclamptic women has been reported recently by Marsh MS, SmithS et al[27.]. This finding suggest that incidence of BAEP becoming abnormal increases with severity of hypertension. This present study concludes that sensory dysfunction of auditory pathway is at the level of brainstem in hypertension.[34]

There is a study Sethi N, Vaney et al [28] reported that decreased sensory conduction during cold pressure response in humans .A few studies showed correlation between metabolic disorders &abnormalities in BAEPs. Some studies says that sensorineural hearing loss with aging is because of microcirculatory insufficiency due to vascular occlusion caused by emboli, haemorrhage, or vasospasm collectively called syndrome of hyperviscosity[15] or microangiopathy caused by hypertension

CONCLUSION

It is concluded that rise in blood pressure affects sensory conduction in the auditory pathway in the brainstem it prolongs the latencies indicating more involvement of 8^{th} Nerve.

So, this study suggests that early screening by Electro Physiological Test may preferably be done to prevent the Hearing impairment at an early stage. It has been proved that BAEP is a valuable method to detect early involvement of auditory pathway. Hence it is considered as useful screening tests for hypertensive subjects. More detail investigations involving BAEP in relation to various diseases have to come up in future.

Future avenues in BAEP:

As the sample size of this study involves small group, further studies are needed in a larger group of individuals to confirm findings.

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ANNEXURE

PROFORMA

Proforma for the study subject : Date:

1.S.NO :

2.Name :

3.I.D (given by investigator) : Contact No.

4.Age :

5.Gender :

6.DiabetologyDept .No. /Master Health checkup no:

7.Occupation : None /Housewife/labourer /professional/others(specify):

8.Religion : : Hindu / Muslim / Christian / Others (specify):

9.Family History of Diabetes : Parents / siblings

10.Educational Qualification : No education /Class I-V /VI – XII /College /Professional:

11.Per Capita Income :

Number of family members :

Net family income :

12.Type of House : Kutcha / Pucca /Semi

13.Amenities at house : Cycle /motorcycle /fridge/electricity/car/mobile/telephone./mobile no:

14.Duration of illness:

15.Social Habits :

	Past/Present/Newer	Brand / quantity	Duration
1.Smoking			
2.Alcohol			

Clinical Details:

16.Symptoms : Duration :

- Increased Appetite:
- Thirst :
- Increased Urination :
- Loss of weight :
- Tingling & Numbness :
- Paresthesia :
- Location :
- Giddiness:
- Skin ailments :
- ENT ailments – Ear Discharge / Wax / Hearing impairment
- Visual Impairment:

17.On Examination :

- Height :
- Weight :
- BMI :
- Blood Pressure :
- Built :

- Nourishment :
- Central Obesity :
- Waist Hip Ratio ;

Final Diagnosis :

Primary Hypertension

Complication :

18.Audiological Examination :

1.Examination of the External Auditory Meatus :

2.Examination of the Tympanic Membrane :

3.Tuning fork tests :

a.Rinnie Test :

b.Weber test :

4.Pure Tone Audiometry :

a.Right ear :

b.Left ear :

20.Brainstem Auditory Evoked Response :

Variables	Right Ear	Left Ear
Latency of waves (ms)		
I		
II		
III		
IV		
V		
Inter-peak latency (ms)		
I - III		
I – V		
III – V		

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.11200/ME-1/Ethics/2013 Dt:12.09.2013
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A evaluation of brainstem auditory response in essential hypertension" - For Research Work Submitted by Dr.V.Priya, MD (Phy), PG Student, KMC, Chennai.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug

INFORMED CONSENT

I undersigned agree to participate in the study entitled ‘’Evaluation of Brainstem Auditory Evoked response in Essential Hypertension’’. All the procedures of the study and possible adverse effects have been explained to me before the test.

Date : Sign of Investigator:

Sign .of Patients :

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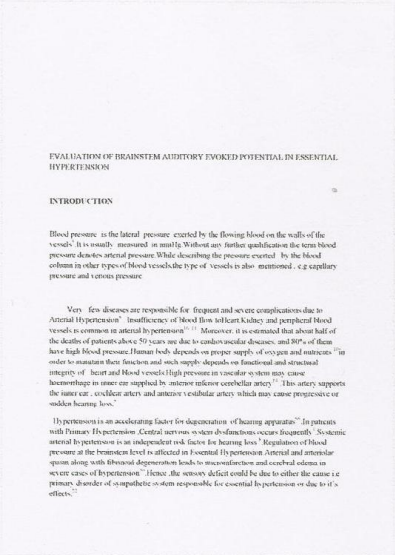


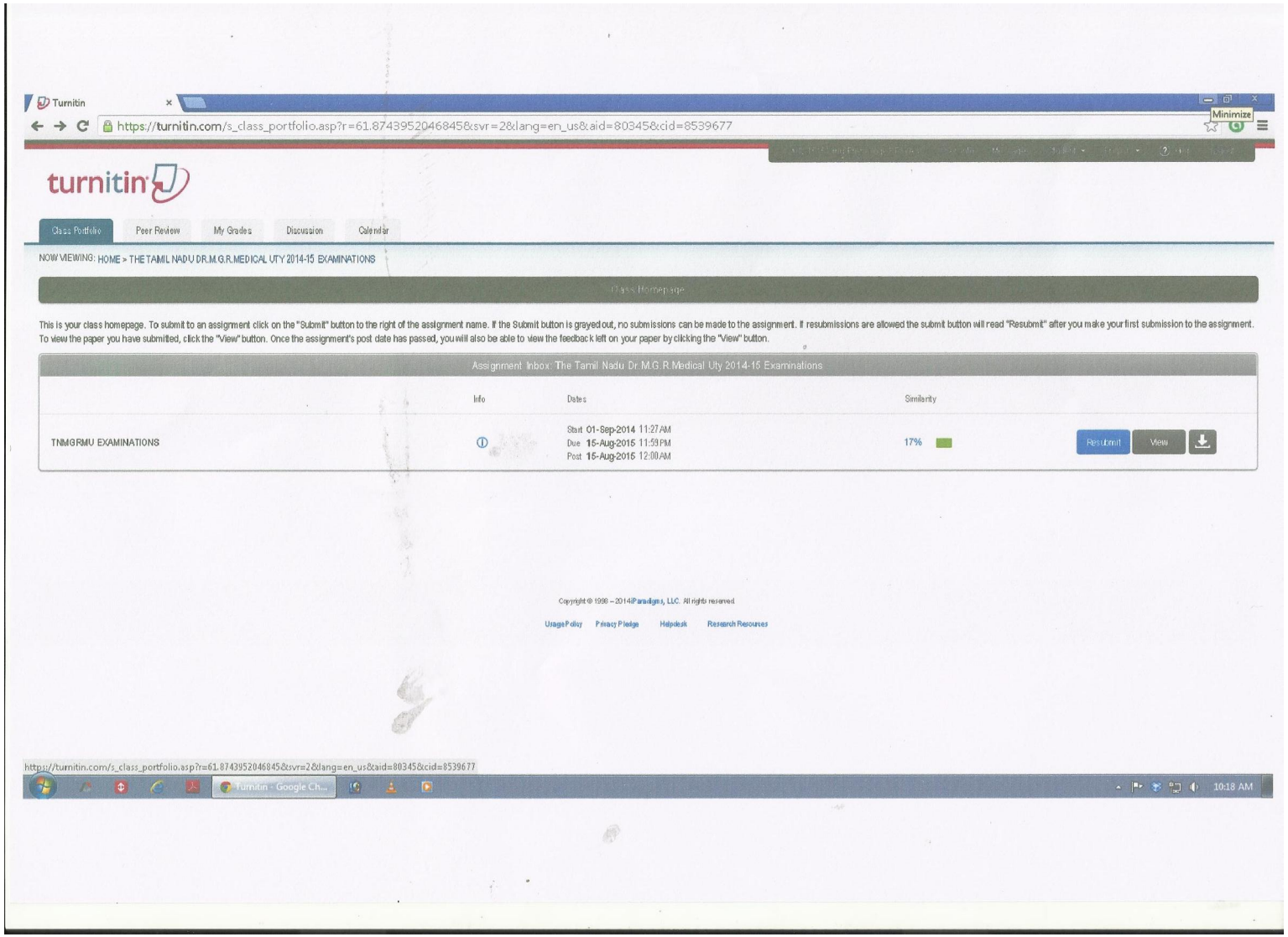
EVALUATION OF BRAINSTEM AUDITORY EVOKED POTENTIAL IN ESSENTIAL HYPERTENSION

INTRODUCTION

Blood pressure is the lateral pressure exerted by the flowing blood on the walls of the vessels[1]. It is usually measured in mmHg.Without any further qualification the term blood pressure denotes arterial pressure.While describing the pressure exerted by the blood column in other types of blood vessels,the type of vessels is also mentioned, e.g capillary pressure and venous pressure

Very few diseases are responsible for frequent and severe complications due to Arterial Hypertension[2]. Insufficiency of blood flow to Heart,Kidney and peripheral blood vessels is common in arterial hypertension[3] [4]. Moreover, it is estimated that about half of the deaths of patients above 50 years are due to cardiovascular diseases, and 80% of them have high blood pressure.Human body depends on proper supply of oxygen and nutrients [5]in order to maintain their function and such supply depends on functional and structural integrity of heart and blood vessels.High pressure in vascular system may cause haemorrhage in inner ear supplied by anterior inferior cerebellar artery[6]. This artery supports the inner ear , cochlear artery and anterior vestibular artery which may cause progressive or sudden hearing loss.[7]

Hypertension is an accelerating factor for degeneration of hearing apparatus[8]. In patients with Primary Hypertension ,Central nervous system dysfunctions occurs frequently .Systemic arterial hypertension is an independent risk factor for hearing loss.[9] Regulation of blood pressure at the brainstem level is affected in Essential Hypertension.Arterial and arteriolar sclerosis along with fibrinoid degeneration leads to microinfarction and cerebral edema in severe cases of hypertension.[10]Hence ,the sensory deficit could be due to either the cause i.e primary disorder of sympathetic system responsible for essential hypertension or due to it's effects.[11]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								LEFT EAR								RIGHT EAR							
S.No	Age	Sex	Height (cm)	Weight(Kg)	BMI	Systolic BP	Diastolic BP	Wave I	Wave II	Wave III	Wave IV	Wave V	IPL I-III	IPL III-V	IPL V-I	Wave I	Wave II	Wave III	Wave IV	Wave V	IPL I-III	IPL III-V	IPL V-I
1	55	M	163	55	20.7	186	118	2.8	3.08	3.38	4.6	5.88	0.58	2.5	3.08	2.38	2.08	2.62	6.2	5.68	0.24	3.06	2.3
2	52	F	158	68	27.24	180	112	2.78	2.58	3.42	6	6.02	0.44	2.6	3.3	1.8	2.3	3.9	5.02	5.5	2.1	1.6	3.7
3	58	M	162	70	26.67	186	118	2.6	2.45	3.48	5.4	5.55	0.88	2.07	1.95	1.65	2.62	3.45	4.53	5.1	1.8	1.55	3.35
4	50	F	158	62	24.84	184	110	2.2	2.72	3.08	5.45	5.98	1.78	2.9	3.78	1.9	2.08	3.05	4.6	5.72	1.15	2.67	3.82
5	46	F	158	63	25.24	182	112	2.05	2.5	3.4	4.95	5.65	1.45	2.25	3.6	1.82	2.6	3.52	4.85	5.8	1.7	2.28	3.98
6	54	F	162	62	23.62	180	110	1.87	2.8	3	5	5	1.13	2.8	3.93	2.84	2.8	3	4.02	5.88	0.16	2.88	3.04
7	57	F	160	65	25.39	184	116	2.9	2.9	3.25	4.72	5.89	0.35	2.64	1.99	2.8	2.38	3	4.75	5.8	1.5	2.8	4
8	55	M	158	60	24.03	180	110	2.88	2.72	3.72	5.08	5.9	0.84	3.08	3.02	1.85	2.98	3.7	5.38	5.82	1.85	2.12	3.87
9	52	M	157	60	24.34	140	90	1.5	1.5	3.3	4	5.1	1.8	1.8	3.5	2.1	2.1	3	4.2	5.6	0.9	2.6	3.6
10	49	M	160	58	22.66	160	104	1.4	2.3	3.3	4	5.1	1.8	1.8	3.6	1.5	2.2	3.3	4.1	5.2	1.9	1.9	3.8
11	55	F	156	68	27.94	146	96	1.4	2.1	2.8	4.3	4.6	1.7	1.8	3.2	1.1	2	3	4.2	5	1.9	1.8	3.9
12	58	F	158	69	27.64	160	106	1	1.98	2.75	4	4.05	1.5	1.3	3.05	1.2	2.2	1.98	4	3.02	0.78	1.04	1.82
13	48	M	162	68	25.91	150	96	1.2	1.8	2.8	4.1	4.7	1.6	1.9	3.5	1.4	2.1	2.8	4.3	4.6	1.7	1.8	3.2
14	52	M	159	61	24.13	166	106	1.1	2	3	4.2	5	1.9	1.8	3.9	1.5	2.3	3.3	4	5.1	1.8	1.8	3.6
15	50	M	162	64	24.39	164	106	1.2	2	3	4	5	1.8	2	3.8	1.2	1.8	2.8	4.1	4.7	1.6	1.9	3.5
16	48	M	155	68	28.3	166	108	1	1.86	2.96	4.2	4.08	1.25	1.12	3.08	1.4	2.1	2.8	4.3	4.6	1.7	1.8	3.2
17	51	F	160	58	22.66	148	96	1	2	2.2	4.1	4.28	1.2	1.9	3.1	1.2	2.2	1.98	4	3.02	0.78	1.04	1.82
18	58	F	158	58	23.23	146	92	1.2	1.8	2.8	4.1	4.7	1.6	1.9	3.5	1	2	2.2	4.1	4.28	1.2	1.9	3.1
19	42	F	158	62	24.84	164	106	1.02	1.62	2.25	3.7	4.2	1.23	1.95	3.18	1.2	1.8	2.8	4.1	4.7	1.6	1.9	3.5
20	59	M	160	68	26.56	160	102	1	2	2.2	4.1	4.28	1.2	1.9	3.1	1.2	2.2	1.98	4	3.02	0.78	1.04	1.82
21	45	M	162	63	24.01	148	98	1.02	1.25	2.02	3	3.2	1	1.18	2.18	1.4	2.1	2.8	4.3	4.6	1.7	1.8	2.2
22	52	F	165	65	23.88	162	104	1.4	1.75	2.2	2.84	3.75	0.8	1.55	2.35	1.02	1.25	2.02	3	3.2	1	1.18	2.18
23	55	M	162	65	24.77	140	92	1.3	1.7	2	2.75	3	0.7	1	1.7	1.02	1.25	2.02	3	3.2	1	1.18	2.18

24	48	M	158	58	23.23	166	108	1.02	1.25	2.02	3	3.2	1	1.18	2.18	1.3	1.7	2	2.75	3	0.7	1	1.7
25	49	F	160	62	24.22	142	92	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6
26	58	M	162	64	24.39	160	100	1.3	2.2	2.8	3.5	4	1.5	1.2	2.7	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6
27	48	F	152	59	25.54	142	92	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7
28	46	M	165	62	22.77	146	92	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
29	58	M	160	60	23.44	162	104	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.25	2.02	3	3.2	1	1.18	2.18
30	60	M	162	58	22.1	146	92	1.3	1.7	2	2.75	3	0.7	1	1.7	1.02	1.25	2.02	3	3.2	1	1.18	2.18
31	48	M	159	60	23.73	166	108	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
32	58	F	160	62	24.22	146	92	1.02	1.25	2.02	3	3.2	1	1.18	2.18	1.3	1.1	2	2.75	3	0.7	1	1.7
33	60	M	158	62	24.84	148	92	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.3	1.1	2	2.75	3	0.7	1	1.7
34	45	F	156	60	24.65	160	100	2.3	2.3	3.3	4	5.1	1.8	1.8	3.6	1.4	2.2	3.3	4.1	5.2	1.9	1.9	3.8
35	55	F	160	62	24.22	162	102	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
36	40	F	158	64	25.64	140	90	1.25	1.75	2	2.75	3	0.75	1	1.75	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
37	59	M	155	60	24.97	162	102	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.25	2.02	3	3.2	1	1.18	2.18
38	48	F	160	62	24.22	146	92	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7
39	55	M	162	65	24.77	162	100	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6
40	45	F	158	60	24.03	146	92	1.02	1.25	2.02	3	3.2	1	1.18	2.18	1.02	1.72	2	2	2.25	0.98	0.98	1.73
41					#DIV/0!																		
42	42	F	158	59	23.63	118	86	1	1.25	1.75	3.2	3.75	0.75	2	2.75	1.2	1.35	1.85	2.75	3.2	0.65	1.35	2
43	48	F	156	60	24.65	128	88	1.3	1.85	2	2.35	2.65	0.7	0.65	1.35	1	1.25	1.75	3.2	3.75	0.75	2	2.75
44	52	M	160	60	23.44	130	86	1.4	1.75	2	2.25	3	0.6	1	1.6	1.25	1.48	2.25	2.5	2.75	1	0.5	1.5
45	55	M	159	62	24.52	120	82	1.2	1.35	1.85	2.75	3.2	0.65	1.35	2	1.3	1.85	2	2.35	2.65	0.7	0.65	1.35
46	48	M	162	58	22.1	128	86	1.45	1.85	2	2.25	2.65	0.55	0.65	1.2	1.65	1.25	1.75	2.25	2.35	0.1	0.6	0.7
47	52	M	165	60	22.04	130	84	1.25	1.45	1.75	2	2.45	0.5	0.7	1.2	1.45	1.85	2	2.25	2.25	0.55	0.65	1.2
48	56	M	160	56	21.88	126	88	1.65	1.25	1.75	2.25	2.35	0.1	0.6	0.7	1.25	1.45	1.75	2	2.45	0.5	0.7	1.2
49	45	F	150	58	25.78	110	70	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6

50	56	F	155	60	24.97	112	70	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
51	50	M	159	61	24.13	120	84	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73	1.02	1.25	2.02	3	3.2	1	1.18	2.18
52	58	F	158	58	23.23	118	82	1	2	2.2	4.1	4.28	1.2	1.9	3.1	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
53	55	M	160	59	23.05	120	88	1	2	2.2	4.1	4.28	0.5	1.9	3.1	1.02	1.72	2	2.25	2.75	0.1	0.75	1.73
54	52	M	158	58	23.23	126	82	1.25	1.45	1.75	2	2.45	1.2	0.7	1.2	1.65	1.25	1.75	2.25	2.35	0.98	0.6	0.7
55	55	M	156	60	24.65	130	88	1.02	1.25	2.02	3	3.2	0.5	0.7	1.2	1.3	2.2	2.45	2.75	3	2.35	0.6	0.7
56	54	M	154	56	23.61	120	80	1.02	1.25	2.02	3	3.2	0.5	0.7	1.2	1.3	2.2	2.45	2.75	3	2.35	0.6	1
57	45	F	152	55	23.81	110	86	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
58	49	F	156	60	24.65	120	80	1.4	2.3	2.25	2.8	3	0.85	0.75	1.6	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7
59	51	F	160	59	23.05	122	88	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.7	2	2.25	2.75	0.98	0.75	1.73
60	57	M	159	60	23.73	126	89	1.02	1.25	2.02	3	3.2	1	1.7	2.18	1.3	2.2	2.45	2.75	3	1.15	1.73	1.7
61	55	M	158	61	24.44	128	90	1	2	2.2	4.1	4.28	1.2	1.18	3.1	1.2	2.2	1.98	4	3.02	0.78	0.55	1.82
62	50	M	155	58	24.14	118	88	1.5	2.3	3.3	4	5.1	1.8	1.9	3.6	1.4	2.2	3.3	4.1	5.2	1.9	1.04	3.8
63	48	M	158	57	22.83	120	90	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
64	50	F	156	62	25.48	126	90	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.25	2.02	3	3.2	1	1.18	2.18
65	60	M	162	64	24.39	130	90	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.4	2.3	2.25	2.85	3	0.8	0.75	1.6
66	56	F	159	60	23.73	130	90	1.3	1.7	2	2.75	3	0.7	1	1.7	1.02	1.25	2.02	3	3.2	1	1.18	2.18
67	47	F	158	60	24.03	126	80	1.02	1.25	2.02	3	3.2	1	1.18	2.18	1.3	1.7	2	2.75	3	0.7	1	1.7
68	50	M	160	62	24.22	128	88	1.4	2.1	2.8	4.3	4.6	1.7	1.8	2.2	1.02	1.25	2.02	3	3.2	1	1.18	2.18
69	45	F	158	60	24.03	128	88	1.2	1.8	2.8	4.1	4.7	1.6	1.9	3.5	1.02	1.62	2.25	3.7	4.2	1.23	1.95	3.18
70	42	F	148	55	25.11	110	70	1.2	2.2	1.98	4	3.02	0.78	1.04	1.82	1.02	2	2.2	4.1	4.28	1.2	1.9	3.1
71	52	M	160	62	24.22	120	90	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.3	1.1	2	2.75	3	0.17	1	1.7
72	50	M	158	58	23.23	126	86	1.3	2.2	2.8	3.5	4	1.5	1.2	2.7	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6
73	52	F	162	63	24.01	130	90	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73
74	58	F	162	58	22.1	130	80	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7
75	50	F	160	62	24.22	110	90	1.3	2.2	2.8	3.5	4	1.5	1.2	2.7	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6

76	49	F	158	58	23.23	120	82	1.3	1.1	2	2.75	3	0.7	1	1.7	1.02	1.25	2.02	3	3.2	1	1.18	2.18
77	52	M	156	58	23.83	120	90	1.65	1.25	1.75	2.25	2.35	0.1	0.6	0.7	1.25	1.45	1.75	2	2.45	0.5	0.7	1.2
78	55	M	160	58	22.66	130	90	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73	1.4	2.3	2.25	2.85	3	0.85	0.75	1.6
79	60	M	158	60	24.03	128	90	1.3	2.2	2.45	2.75	3	1.15	0.55	1.7	1.02	1.25	2.02	3	3.2	1	1.18	2.18
80	57	M	159	59	23.34	120	86	1.25	1.75	2	2.75	3	0.75	1	1.75	1.02	1.72	2	2.25	2.75	0.98	0.75	1.73